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MORBIDITY AND MORTALITY IN THE CONVICT SETTLEMENT AT PORT ARTHUR, TASMANIA, FROM 1830 TO 1835.[1]

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ON a visit a few years ago to that most interesting place, the old convict settlement at Port Arthur, in Tasman's Peninsula, Tasmania, I had an opportunity of seeing the beautifully written official manuscript return, then in the possession of Mrs. Barnett at Port Arthur. This return is headed: "Tasman's Peninsula, Van Dieman's Land: Return of Various Particulars Relative to the Penal Settlement formed at Port Arthur from 9th September, 1830, to 31st December, 1835." In the "Handbook to Tasmania", Australasian Association for the Advancement of Science, Hobart, 1928, it is stated that Port Arthur was founded by Dr. Russell on September 22, 1830.

Amongst the particulars enumerated are the quantity and quality of the work done by prisoners, with its estimated value, certain diet scales *et cetera*, and, of special interest to medical men, returns giving the diseases and the deaths during the period under review.

These returns show that the numbers of convicts actually on the strength at the termination of each year were as follows: 1830, 68; 1831, 151; 1832, 278; 1833, 475; 1834, 735; 1835, 911. The total number of convicts received during this period was 1,711; of these 723 were sent up to Hobart for good conduct; four men were drowned in a boat in the harbour in 1832; and 68 additional deaths are recorded for this period in the table accompanying this note.

The following is an abstract of those items of more particular interest at the present day, entered under the title "Return of Diseases and Showing the

[1] Read at a meeting of the Section of the History of Medicine of the South Australian Branch of the British Medical Association on June 23, 1932.

Number of Cases Treated in His Majesty's Colonial Hospital from 9th September, 1830, to 31st December, 1835, inclusively". (Note that the year of the first item of each disease is inserted in brackets and then follow the number of cases of that disease for that year and the consecutive years, up to 1835, or as many years afterwards as showed the incidence of this disease.)

Ambustio: (1831) 15, 2, 22, 7, 5. (Total, 51.)
Abscessus: (1830) 7, 45, 25, 31, 6, 26. (Total, 140.)
Ascarides: (1835) 2. (Total, 2.)
Cynanche: (1831) 6, 3, 31, 35, 9. (Total, 84.)
Cholera: (1834) 16. (Total, 16.)
Diarrhœa: (1830) 6, 13, 45, 232, 386, 289. (Total, 971.)
Dysenteria: (1830) 2, 40, 20, 93, 24 (7 deaths), 135 (9 deaths). (Total, 314.)
Eruptio: (1832) 13, 34, 52, 14. (Total, 113.)
Erysipelus: (1831) 1, 0, 4, 13, 21. (Total, 39.)
Febris: (1830) 4, 15, 8, 29, 54 (2 deaths), 122 (3 deaths). (Total, 232.)
Furunculus: (1833) 123, 69, 35. (Total, 227.)
Gonorrhœa: (1833) 6, 6, 2. (Total, 14.)
Hæmoptysis: (1833) 10, 6, 5 (2 deaths). (Total, 21.)
Herpes: (1830) 1, 0, 0, 12. (Total, 13.)
Icterus: (1835) 7. (Total, 7.)
Laryngitis: (1835) 5. (Total, 5.)
Morbus cordis: (1833) 4 (1 death), 9, 18. (Total, 31.)
Nephritis: (1835) 2. (Total, 2.)
Obstipatio: (1830) 6, 43, 16, 123, 224, 139. (Total, 551.)
Odontalgia: (1830) 2, 13, 3, 50, 41, 37. (Total, 146.)
Dentitis: (1830) 1. (Total, 1.)
Ophthalmia: (1830) 1, 3, 0, 36, 62, 64. (Total, 166.)
Otitis: (1833) 5, 11, 2. (Total, 18.)
Paronychia: (1831) 2, 0, 23, 18, 39. (Total, 82.)
Partus: (1833) 1, 2, 8. (Total, 11.)
Pediculi: (1834) 5, 3. (Total, 8.)
Pernio: (1835) 12. (Total, 12.)
Phlegmon: (1830) 7, 46, 24, 0, 102, 81. (Total, 260.)
Pleuritis: (1833) 5, 30, 25. (Total, 60.)
Pneumonia: (1831) 20, 1, 13, 27 (1 death), 5 (2 deaths). (Total, 66.)
Punitas: (1830) 1, 0, 0, 53, 46, 23. (Total, 123.)
Phthisis: (1834) 1 (1 death). (Total, 1.)
Rheumatismus: (1831) 16, 5, 126, 207, 79 (2 deaths). (Total, 433.)
Rubeola: (1835) 1. (Total, 1.)
Scabies: (1831) 10, 0, 1, 3, 2. (Total, 16.)
Scorbutus: (1830) 7, 203, 46, 321, 412 (1 death), 26. (Total, 1,015.)
Subluxatio: (1830) 1, 13, 10, 88, 17, 9. (Total, 138.)
Struma: (1835) 2. (Total, 2.)
Testitis: (1831) 1, 1, 10, 7, 5. (Total, 24.)
Tumor: (1834) 8. (Total, 8.)
Ulcus: (1830) 3, 55, 25, 32, 32, 47. (Total, 194.)
Urticaria: (1835) 4. (Total, 4.)
Vermes: (1830) 1, 4, 2, 22, 43, 18. (Total, 90.)
Vertigo: (1831) 12, 29, 103, 63, 11. (Total, 218.)
Vulnus: (1831) 42, 20, 84 (2 deaths), 83, 56 (1 death). (Total, 285.)
Patients treated: (1830) 69, 832, 408, 2,235 (7 deaths), 3,067 (27 deaths), 2,040 (34 deaths). (Total, 8,651, with 68 deaths.)

The following is a list of deaths during the period under review: Anasarca, 2; *apoplexia*, 1; bronchitis, 1; cephalagia, 1; cerebritis, 5; dysentery, 16, dyspepsia, 2; empyema, 2; *debilitas*, 2; *febris*, 5; fracture, 4; gastritis, 4; gastro-enteritis, 3; hepatitis, 2; hæmoptysis, 2; *morbus cordis*, 1; pericarditis, 1; peritonitis, 2; phrenitis, 2; pneumonia, 3; *rheumatismus*, 2; *scorbutus*, 1; *vulnus*, 3; phthisis, 1.

In studying these returns it is necessary to remember that the figures for 1830 deal only with less than four complete months, with a total of approximately 68 convicts, and that the numbers (at the end of each year) increased as follows: 151, 278, 475, 735 and 911 (in 1835). As these figures refer to the number in the establishment at the end of each year, the actual number of persons liable to sickness during each year must be somewhat greater, this allowing for those who were returned to Hobart (723 in all) and those who died (68).

The greatest invalidity was due to scurvy (*scorbutus*) with a total of 1,015 treatments out of 8,651, though only one death resulted. There seems no need to question the diagnosis, the disease being then widely known and readily recognizable.

It is interesting to note that James Backhouse,[1] the Quaker, was present in Tasmania during part of the period under review, and actually made three visits to Port Arthur, with rather significant results. His first visit was towards the end of September, 1832. He states that:

> Allowing such of the prisoners as conducted themselves well at this place, a few square yards of ground for gardens, and a small quantity of tea and sugar, had a very beneficial influence upon their conduct, but these indulgencies being considered incompatible with the rigid nature of the discipline intended to be maintained at such stations, were after a time withdrawn.

If now we refer to the patients with scurvy admitted to the hospital, it will be seen that there were a large number of cases in 1831, namely, 203, and that in 1832 there were only 46; evidently this considerable drop was due to the practice referred to by Backhouse of allowing the better behaved prisoners small patches of ground for gardens. The withdrawal of this privilege apparently is reflected in the scurvy returns. They increased in 1833 to 321, this being the year following Backhouse's first visit. His second visit was in November, 1833, Captain Charles O'Hara Booth being then in command. In 1834 there were 412 cases and one death, but in 1835 they had dropped to 26. The explanation of this sudden drop is not hard to find. We find that the excellent Backhouse was presumably responsible. Backhouse paid a third visit in October, 1834. He found the settlement greatly improved since his former visits. The number of prisoners was then 887. He writes:

> In consequence of the prisoners living on salt meat and being defectively supplied with vegetables, a large number were suffering from scurvy; 19 were in the hospital chiefly from this disease, and presented as appalling a picture of human wretchedness as I recollect ever to have witnessed; this defect, on being represented to the Government, was speedily remedied by the cultivation of some land with vegetables and an occasional supply of fresh meat.

Backhouse also visited Point Puer, where 157 boys were under restraint. He said the restraint was irksome, but, upon the whole, the boys seemed pleased with the idea of being taught work that would enable them to maintain themselves later. There is no suggestion that these boys were unduly harshly treated at this period. One knows of the appalling stories that have been told of ill-treatment at this place.

Ambustio is presumably "burns" (Latin *amburo*, to scorch, to burn wholly up). Fifty-one cases seems a large number, mostly contracted in the second year (15) and the fourth (22). Possibly scrub was

being burnt off and, if the convicts were working in chains (as would be likely from the ease of escape in the dense undergrowth), their agility would be lessened and escape from rapidly spreading flames less easy.

Abscesses (140), erysipelas (39), *furunculus* (227), paronychia (82), phlegmon (260) and ulcers (*ulcus*) (194) all loom largely and may be taken together as being pyogenic infections by streptococci or *Staphylococcus aureus*, perhaps predisposed to by a diet deficient in vitamin *A* (as is quite likely to have been the case). If these are added up, we find that the totals for the years 1830 to 1835 were respectively as follows: 17, 148, 75, 213, 240 and 249, a total of 942 cases. We see a distinct drop, as in scurvy, for 1832, but no final drop in 1835, in spite of Backhouse's recommendation and its result. Furunculosis appears only for the last three years, so perhaps the diagnosis entered on the records may have varied with the personnel or the whims of the medical officers concerned. Thus one man might have preferred to call boils abscesses, and another to call certain abscesses boils. To some extent, as shown in the accompanying table, the figures under the different headings vary in the same direction for the respective years.

Condition.	Year.					
	1830	1831	1832	1833	1834	1835
Abscesses	7	45	25	31	6	26
Erysipelas	—	—	1	4	13	21
Furunculus	—	—	—	123	69	35
Paronychia	—	2	—	23	18	39
Phlegmon	7	46	24	—	102	81
Ulcers	3	55	25	32	32	47
Totals[1]	17	148	75	213	240	249

[1] The total for all the years is 942.

Of parasites, two cases of ascaris infection, 90 of vermes infestations (perhaps mostly threadworms), eight of pediculosis, and 16 of scabies are recorded. The latter two headings doubtless represent aggravated infestations, light infections being probably considered as almost inseparable from convict assemblages. Cynanche (from the Greek κύων, κυνός, a dog, and ἀγχόνη, throttling, strangling) means a swelling in the throat, especially quinsy. Of this there were 84 cases. This disease probably should be placed with the pyogenic infection group.

Cholera (16 cases, all in 1834), diarrhœa (971) and dysentery (314 with 16 deaths) may all be taken together. The diagnosis of cholera cannot be seriously entertained. The disease has never established itself in Australia, though many years ago a ship arrived at Brisbane with true cholera aboard and was quarantined at Dunwich, on Stradbroke Island, in Moreton Bay. "Dysentery" also may merely have been a severe diarrhœa from unsuitable food, or, on the other hand, some cases both of this and of the other two headings may have been true bacillary or even amœbic dysentery. The figure 24 (with seven deaths) under "dysentery" for 1834 is probably too low and due perhaps to an error in transcription on my part. Both diarrhœa and dysentery increased in prevalence in the last three years.

What the 113 cases of *eruptio* with a spread over four years were I do not know. Perhaps they were a miscellaneous collection of skin diseases. Measles (rubeola) appears as a single case in 1835. Being alone, the diagnosis was doubtless at fault. The "eruptions" seem not to have been an exanthem, as if so, surely one or more deaths would have occurred.

The 232 cases of fever with only five deaths does not suggest typhus, though some cases cannot perhaps be excluded. It may be considered as a conglomeration of illnesses in which continued fever occurred. Typhoid fever may have been amongst them, but "colonial fever", as typhoid was first called, appeared recognizably only after gold "broke out" in the fifties.

There were 14 cases of gonorrhœa, but there is no mention of syphilis (or "the pox") or its aftermaths. There were 24 cases of testitis spread over five years, and they were probably gonorrhœal and not due to mumps.

Twenty-one patients had hæmoptysis, of whom two died, and another died of phthisis. These were probably mostly, if not all, cases of pulmonary tuberculosis. Struma appears as two cases.

A mild outbreak of herpes, perhaps *herpes zoster*, appears to have occurred in 1833 (12 cases out of the 13). There were 31 cases of heart disease (probably of very varied nature) with only one death, two of nephritis, 60 (a high figure) of pleurisy (probably including some pneumonias), 66 of pneumonia (with three deaths) and 433 of rheumatism, with two deaths. The last is a vague term, embracing doubtless acute rheumatic fever, rheumatoid arthritis, osteoarthritis, gonorrhœal arthritis, and perhaps other conditions with aches and pains in the joints. The invalidity under this heading was great. *Obstipatio* is an old name for constipation, from which 551 of the patients suffered. It is not likely that many, if any, patients were catalogued under more than one heading, so these were doubtless all "out-patients".

Toothache accounted for 146 patients, but dentitis (whatever this means; perhaps the gums of scurvy) only for one. Inflammation of the eyes (ophthalmia) totalled 166, and otitis 18.

Partus must mean childbirth, and the eleven patients treated were presumably the wives of the staff.

Pernio (chilblains), 12 cases, seems a trivial entry.

Punitas, 123 cases, evidently refers to the after-effects of floggings and possibly other results of severe punishments.

If *subluxatio* means dislocations, the figure 138 is extraordinarily high, even if the phalanges are included. Eighty-eight occurred in one year (1833).

Perhaps clearing dense forest areas led to many minor injuries of this nature.

Only eight tumours were recorded, with no deaths. Were malignant growths rare at this period? Most of the convicts sent to Port Arthur would be in the full flush of manhood and so below the cancer age.

Two hundred and eighteen patients suffered from vertigo, but none died. Cerebral hæmorrhage, then, is clearly not included, and after all, the men were mostly too young for this. The cases of vertigo or giddiness and fainting were most numerous in the two years when scurvy claimed most victims, and this is a symptom in severe cases of this disease. But if so, would the patients not have appeared under *scorbutus* instead of vertigo? They would not be likely to appear under both.

Wounds (*vulnus*) account for 285, most probably due to the dangers, especially to men in chains, encountered in felling trees and clearing scrub. Backhouse gives an illustration showing a chain gang going to work near Sydney. The unfortunate felons, carrying spades, picks, hammers and so on, have each a chain round the loins with a stem descending in front till opposite the knees, when it bifurcates into a chain of links descending to each ankle, to be attached to an anklet. Most of the men are depicted as holding the chain up a little in front of them to prevent the linked part from dragging round the ankles. Some wounds may be accounted for by gunshot injuries received when attempting to escape.

James Backhouse also visited the notorious penal settlement at Macquarie Harbour in 1832. He has a few remarks to make of medical interest in this connexion. Out of 85 deaths that had occurred there in eleven years, commencing with 1822, only 35 had been due to natural causes. Of the remainder, 27 were caused by drowning, eight men were killed accidentally, chiefly by the falling of trees, three had been shot by the military, and 12 murdered by their comrades. Backhouse said that there was reason to believe that some of these murders had actually been committed for the purpose of obtaining for the murderers and the witnesses a removal from Macquarie Harbour to Hobart, even though it meant for the murderer death in the end.

During the eleven years 112 prisoners had escaped, 62 of whom were supposed to have perished in the bush and nine to have been murdered by their comrades and eaten. One party, later apprehended, had in their possession portion of one of their comrades as well as the flesh of a kangaroo.

During the three years 1826, 1827 and 1828, 188 prisoners out of 312 had been punished, 6,280 lashes in all having been inflicted upon them. During 1829, 1830 and 1831 only 56 prisoners out of 255 had been sentenced, the lashes inflicted falling to 973. The general health at Macquarie Harbour was good, seldom more than three being in hospital at a time. The average of deaths did not amount to more than one in 35 *per annum*, including those by violence and accident. Backhouse attributed the relative freedom from disease, especially in men whose habits had been dissipated, to the spare diet and hard labour in the hot though humid climate, where they were prevented from having access to strong drink. He adds, however, that the surgeon remarked that when the men did become ill, the tone of their constitution was so low that it was difficult for them to recover. Backhouse thought this might be due to the limited supply of food or from being restricted to the use of salt meat.

As giving a further insight into the diseases prevalent at this time in Tasmania, a work published in 1833 by H. W. Parker,[2] a barrister, may be quoted. He mentions that a year or two before this date "a kind of influenza, a cold and slight fever" was prevalent, but had not been attended with danger. Fever and dysentery were the most common complaints. Whooping cough had appeared within the last half dozen years and was said to have been introduced by female convicts; notwithstanding that it had spread almost throughout the whole population, no deaths occurred. Temporary insanity, both among male and female prisoners, occurred more frequently than was usual in England. Several instances of goître had appeared in particular localities, but none had been severe. He adds that goître is a complaint incidental to most mountainous countries and might therefore be expected to be found in an island where the surface was so very unequal.

References.

(1) James Backhouse: "A Narrative of a Visit to the Australian Colonies", 1843.
(2) Henry Walker Parker: "The Rise, Progress and Present State of Van Diemen's Land", 1833, pages 74, 75.

SINUSITIS AND TUBERCULOUS INFECTION.

By SYDNEY PERN, M.R.C.S. (England), L.R.C.P. (London), *Melbourne.*

THE method by which the tubercle bacillus gains entrance to the lungs is still a matter of dispute. There are many who believe that it is carried by the air into the alveolus; others think that it first gains entrance to a gland and is then conveyed to the alveolar tissue by the lymphatics or blood stream. That tubercle bacilli, except in overwhelming doses, can be carried by the air to the alveolus seems improbable, as there are a hundred and one traps on the way. Long before it gets near the alveolus a tubercle bacillus must impinge upon some mucous membrane, the ciliary epithelium of which would sweep it out again. One has also to bear in mind that the residual air in the alveoli is not discharged during expiration, only an interchange of gases taking place.

For some years past the writer has been immensely struck by the amount of hilar thickening seen on the screen and radiographic films in patients suffering from nasal infection. This hilar thickening is indistinguishable from that of a tuberculous origin or of that seen in cases of undoubted pul-

monary tuberculosis. The thickening itself is here referred to and not the association of calcareous glands and other signs on the film. This association of hilar thickening and nasal infection is gradually becoming more recognized.

The presence of nasal infection and its association with disease in general is only just beginning to be realized. It was dealt with in a paper by the writer in 1925. This infection is so common that 25% of people attending medical clinics, as judged by my own, have this infection. The *sine qua non* of infection is the presence of a chronic mucopurulent post-nasal drip accompanied by the usual signs of catarrh. The hilar thickening of the lungs is obviously lymphatic in origin. The nasal infection, either in itself due to faulty drainage of sinuses or inducing faulty drainage due to swelling of the mucosa, tends to an invasion of organisms which are carried *viâ* the lymphatics down the posterior chain of glands behind the sterno-mastoid muscle to the mediastinal glands, and from there are relayed along the lymphatics embracing the bronchial tubes. In this way chronic bronchitis is gradually induced. There are some of the medical profession who for long have recognized that an alveolitis not of tuberculous origin was possible, and in the presence of marked nasal infection with absence of tubercle bacilli on repeated examinations, the writer has been reluctant to pronounce these patients tuberculous, especially in the absence of any history of close association with open tuberculosis. If 25% of people attending medical clinics have nasal infection, questions urgently requiring investigation are what proportion of the community at large is so infected, what bearing this infection has in helping the conveyance of the tubercle bacillus to the lungs, and how far it affects the course of the disease in tuberculous patients in general. One can hardly conceive that a continual stream of bacteria passing from the nasal mucosa to the bronchial lymphatics can do anything but lower the resistance of the individual and considerably hinder him in overcoming a tuberculous infection.

Three years ago, a girl of twenty-two years presented herself for treatment with enlarged tuberculous glands along the posterior borders of each sterno-mastoid muscle. One of these glands was already becoming soft. She had obvious nasal infection. She was given X ray therapy and her nasal condition was treated. In spite of trying to empty the contents of the broken-down gland by a hypodermic needle, the gland ulcerated and had to be dealt with by ultra-violet rays. This speedily brought about healing. Her nasal condition cleared up and all enlargement of glands vanished.

A month or two ago she came again with the same glands enlarged and a return of the nasal trouble. X ray treatment was given, with the same treatment for her nasal condition, but this time without success. As the condition of the glands refused to improve, she had radical operations on her antra, and without any further treatment the infected glands became normal. Her father suffers from pulmonary tuberculosis.

In October, 1927, another girl, aged twenty-one years, came with a similar condition. A broken down gland was aspirated and treated with X rays. The glandular enlargement disappeared in about a month, but returned eighteen months later. Aspiration was carried out on several occasions and her nasal condition was attended to. She has had no return of the trouble during the last two and a half years.

The relationship between tuberculous infection of glands of the neck posterior to the sterno-mastoid muscle and nasal infection strongly suggests that the tubercle bacilli were gaining entrance through the nasal mucosa and probably through the mucosa lining a sinus. If this is so, it is highly probable that the enlarged glands were infected from this source and not from a casual infection, but from bacilli incubating in an infected sinus. It is of great importance that this question be settled, as there does not appear to be any available literature dealing with it.

One cannot conceive that organisms can travel against the lymphatic flow, but one can readily see that, once they are in the lymphatic system, they can easily be conveyed to other parts of the body. At the same time the spread of hilar thickening is from the centre to the periphery and is probably by direct continuity against stream. The only other possibility is by direct entrance of the bacilli into a small vessel or by their gaining entrance to a vessel by means of a leucocyte. In this way it would quickly reach the periphery of the lungs. Another point worthy of consideration is how much of the hilar thickening in tuberculous infections is due to the tubercle bacillus and how much to other organisms. In either case there has to be an inflammatory process to start with.

A patient of fifty-three years, who has nursed many tuberculous patients, comes with asthma. She has a tremendous amount of fibrosis of the lungs with a good deal of frothy expectoration. The X ray film was not very helpful. No tubercle bacilli could be found. She is very thin and is still losing weight. She has had nasal infection for years. Her condition suggests an old standing case of fibroid phthisis.

In this case, which caused the fibrosis: the tubercle bacillus (if it was present) or the organisms from the nasal infection? Whilst attributing most of these hilar conditions to nasal infections, one must not lose sight of the possibility of tonsillar infection causing the same thing, although it causes enlargement of a different chain of glands.

It is very well known that a great deal of the toxæmia of pulmonary tuberculosis is due to secondary infection, and if this is coming from the nasal sinuses, one might hope that great benefit would be derived from clearing up this source of trouble in the early stages. In a large number of cases this can be accomplished by the simple process of giving half a dozen injections of one cubic centimetre of colloidal manganese, one injection twice a week. The question of nasal operation in tuberculous conditions has to be weighed in each individual case. The same can be said of tonsillar infection, though the diathermy method of destroying tonsils at four or six sittings causes very little upset and could be readily undertaken where the upset from enucleation might be feared. This fear is well warranted, as catastrophes such as the development of miliary

tuberculosis occasionally occur after enucleation. Here there is a large raw surface directly infected with quantities of tubercle bacilli expressed from the crypts. On the other hand, when the diathermy needle is used, the part touched is destroyed and sterilized, whilst the underlying area is not interfered with. Statistics show that tuberculous infection found in tonsils which have been enucleated varies from 2·35% to 5·0%. The appearance of an infected tonsil associated with tuberculous adenitis in no way differs from that of an ordinarily infected tonsil. For tuberculous adenitis to develop it would appear necessary that tubercle bacilli were multiplying in some of the crypts without causing any direct tuberculous disease of the tonsil itself, rather than that a few stray bacilli had gained entrance to the tonsil and had been passed on to the gland. If this be so, the same condition may pertain in the nasal sinuses without producing any definite tuberculosis of the nose. If such a condition could be shown to exist, it would have a very important bearing on future treatment, because if this is so and this breeding ground be left alone, it becomes a constant source of infection to the individual.

It is quite obvious that bacteria bred in tonsils gain entrance to the blood stream and are carried to different parts without any flare-up necessarily taking place in the tonsils themselves. What is there to prevent tubercle bacilli from being incubated in some sinus or tonsillar crypt and finding their way likewise into the blood stream without showing evidence of their presence in such a sinus or crypt? Gonococci do the same thing elsewhere. It is improbable that tubercle bacilli would find their way as freely through a healthy mucosa as they would through one already infected with organisms, but if sinus infection is already present, with leucocytes carrying streptococci into the blood stream, what is there to prevent them carrying a few tubercle bacilli if they are present also? It would thus seem that the presence of sinusitis would be a potent factor in the invasion of the body by the tubercle bacillus, especially if one bears in mind that whilst in a sinus or tonsillar crypt, the tubercle bacilli are immune from the attacks of the defence mechanisms of the body, whereas, once within the tissues, they are immediately assailed.

That a tubercle bacillus could reach the neighbourhood of the apex of the lungs *viâ* the lymphatic stream requires a good deal of imagination, but that it should get there *viâ* the blood stream is more or less possible. If we trace an organism entering the body through an abrasion, it is the lymphatics that bear the brunt of the primary attack. Red lines run up to the nearest glands, where every effort is made to hold up the invading force. Once past these, travelling along the thoracic duct, the organisms enter the subclavian vein and so pass into the heart and through the lungs. It is at this stage that tubercle bacilli or other organisms are likely to be held up, where the stream is most sluggish.

If, on entering the blood stream, they are able to withstand the bactericidal forces of the blood, they are passed on to be dealt with by the kidneys, spleen and liver.

We must realize that bacteria incubating within a tonsillar crypt or sinus are immune from destruction on the part of the body's defence forces as long as they do not penetrate the tissues. How they gain entrance into blood or lymphatic stream is the question. Apparently the most probable method of entry is by means of leucocytes, and as infected tonsils and antra are often, but not always, accompanied by enlarged glands, one assumes that the organisms are carried into the lymphatic stream and after running the gauntlet, into the blood stream. When tonsils are red and inflamed, it is probable that bacteria are multiplying within the tonsillar tissue itself as well as in the crypts; but one often sees pale-looking tonsils from the crypts of which infected material can be expressed, suggesting that there is little reaction taking place within the tonsillar tissue itself. The existence of infective conditions which are undoubtedly of tonsillar origin and in which there is no glandular enlargement, is suggestive of bacteria directly entering the blood stream, although on the whole the chances are in favour of them having to run the gauntlet of the lymphatic system first.

If we apply this same view to air-borne infections, such as scarlet fever, diphtheria *et cetera*, in which the point of entrance is the nasopharynx, it is obvious that in the presence of other infections the path of entry to the blood stream is much facilitated, and with the added infection there is a slump in the resistance of the body, allowing the original infection to run riot. There is also the question of symbiosis to be considered. We know that certain organisms thrive better in relationship with others of a different species. In recent work done by Dr. Kijujewa and Dr. Kaschajewa, of the North Caucasian State Microbiological Institute, it was found that the influenza bacillus grew best in symbiotic relation with *Streptococcus brevis* and with pneumococci.

Take three examples. A girl has an enlarged gland under her jaw, an infected tooth is removed, the glandular enlargement disappears. A child has enlarged submaxillary and cervical glands, infected tonsils are removed, the glands vanish. In this type of case one cannot be sure whether the glands are tuberculous or not unless they caseate. Even when they are definitely tuberculous, removal of the tonsils, as far as my experience goes, has been sufficient to cause resolution of those glands which have not broken down. A girl has a radical operation on her antra. From that time she has no recurrence of the tuberculous enlargement of the glands in her neck. The consideration of these facts points to the possibility of tubercle bacilli or other organisms growing in some place outside the body tissues, such as in the antra and crypts of the tonsil and passing uninterruptedly into the body, probably through the agency of leucocytes. If other

infection is also present in these localities, this infection will pave the way for the entrance of the tubercle bacillus, owing to the inflammatory reaction already set up. If this inflammatory reaction were not present, it is possible that the tubercle bacillus might not gain entrance at all. It would seem that the lymph glands have a very high bactericidal action and that their persistent enlargement is due to the fact that they are continuously receiving fresh supplies of organisms, and that suppuration and caseation occur only when the supplies come in overwhelming amounts. If this was not so, the glands would not subside on removal of the tonsils or of the lining of the antra. It is also obvious that a time may come when foci form in various parts of the body and immunity is lost, resulting in scattered glandular enlargement. The fact that tubercle bacilli are incubating within an antrum or tonsillar crypt and not leading to any pathological changes in the walls of those structures makes it difficult for us to be sure of their presence.

It is definitely known that tubercle bacilli do live in tonsils, and it is also obvious that they must be constantly gaining an entrance to the blood and lymphatic streams from that source. How many children are there with tuberculous bones and joints with tubercle bacilli in their tonsils becoming reinfected every day from that source? If we compare the casual infection when inhaling tubercle bacilli or swallowing them in our foods with the daily infection arising from their entrance through the tonsils, it is quite obvious that the one is a thousand times more potent than the other, and if it were not for the efficient guardianship of the glands, the individual would soon be overwhelmed. If this is a fact, as is suggested here, and there seems no doubt of the truth of it, the removal of this source of daily infection is going to advance the results of treatment materially, and once this point is thoroughly understood, it will have a great effect in diminishing the incidence of tuberculosis.

Carrying these views in our mind it is at once obvious that some research bearing on these lines might throw further light upon the subject. Without having made any statistical study as to the relationship between sinusitis and pulmonary tuberculosis, the writer has been struck with the fact that the patients seen by him during the last year or two have had nasal infection.

Whilst discussing this question of lung infection and its association with sinusitis, one has also to consider the relationship of silicosis and pneumonokoniosis with this condition, for it must go without saying that a man with chronically infected tubes will be less able to discharge dust particles than a normal man, and as the infection increases the ciliary epithelium will lose its function. This naturally opens up the very important question of making a special nasal examination of all people entering upon trades in which dust and silica are likely to be encountered, with a view to excluding from such employment all those found to be infected.

SPONTANEOUS SUBARACHNOID HÆMORRHAGE.

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THE recognition of spontaneous subarachnoid hæmorrhage as a clinical entity is comparatively recent. Wilks described in 1859 the autopsy findings in four cases of subarachnoid hæmorrhage not due to accident or related to intraventricular bleeding. Interest in the topic was only languidly maintained until the advent of lumbar puncture, but in the last fifteen years much literature has accumulated. The commonest causes of this vascular accident are arterial degeneration in the older patients, and either congenital aneurysms or congenital defects in the cerebral vessels in those of earlier age.

The practitioner of medicine is but faintly interested in those intriguing but uncommon curiosities, the larger intracranial aneurysms, but he should be alive to the fact that small defects or actual sacs may exist in or be acquired by the young as well as the old. Moreover, he should remember that leakage gives rise to recognizable symptoms in many cases, and that rupture, allowing the escape of blood under the arachnoid, gives rise to a clinical syndrome that is dramatic, perfectly clear-cut and well defined. Since more than half the sufferers from this condition recover, the matter is literally of vital interest.

Symptoms.

The symptoms may vary in intensity from a mere attack of giddiness with headache (which, of course, could hardly be traced to its true cause) to a sudden apoplectic seizure, with swiftly lethal result. But between these extremes lies the more usual type, in which, after a sudden onset with headache and a variable degree of coma, a picture of definite meningeal irritation is seen, and the lumbar tap reveals bright blood-stained fluid. It will probably be most useful to detail the signs and symptoms serially.

The onset is usually rapid. If the leak is slight and slow, little may be felt but pain and stiffness in the back of the neck, with headache. Indeed, repeated migrainoid attacks have been ascribed to such a cause when aneurysm was afterwards found to exist. In the severer cases the patient may call out with pain in the head and rapidly collapse, soon becoming comatose.

Headache is invariable. It is frequently occipital and accompanied by neck stiffness. It may occasionally be the only symptom, for a time at least, as in an interesting case reported by Collier.[1] In this instance a woman drove a motor car from Liverpool to London, though suffering from intense headache; she presented no abnormality on physical examination, but she died suddenly eight hours later, and lumbar puncture, carried out after death, revealed blood under high pressure.

Neck rigidity is a sign of meningeal irritation and is always present to some degree. It may appear early or only after the first signs of shock have abated. Its importance lies in the fact that in a young person in particular it suggests meningitis. Kernig's sign is usually present and well marked. In conjunction with this may be noted the patient's attitude, one of general flexion; this posture may be maintained for days.

Fever is quite common and may persist even after the cerebro-spinal fluid no longer contains blood cells. In combination with the above signs it may strongly suggest a meningeal infection, but the nature of the spinal fluid reveals the true state of affairs.

Coma occurs in all severe cases. It depends upon the degree of intracranial pressure and does not preclude recovery. Patients may recover after deep coma and days of subsequent cerebral irritability and confusion. A lethargy resembling that of encephalitis may also occur.

Mental confusion is also a common symptom and occurs during the recovery period. The Korsakoff syndrome of the alcoholic is sometimes seen. The patient is usually extremely irritable.

The cerebro-spinal fluid is under increased pressure and always contains blood intimately admixed with it. The amount of blood is at times surprising; a count of over three million red cells per cubic millimetre has been observed. Certainly it comes as a shock to the medical attendant to see bright blood-stained fluid gush from the needle if he does not previously suspect the nature of the condition. Probably he fears that he may have caused some traumatic bleeding, but this is easily decided. The blood is evenly mixed, and fluid collected in serial test tubes maintains an equal degree of colour; it does not clot, and when it is allowed to settle or when it is centrifuged, a yellow colour appears in the supernatant fluid, whereas traumatic blood stains the fluid not at all after spinning. After a variable period the blood disappears, though the fluid may be xanthochromic for some time afterwards. An excess of leucocytes is found at this time also. This is not due, however, to infection, but simply to the persistence of the white blood cells after the reds have been disintegrated.

In regard to localizing signs, slow leaks may give rise to misleading symptoms suggesting some other lesion, such as tumour, unless the indication of the lumbar puncture findings be observed. Diplopia during the attack, or a subsequent eye palsy, may help to localize the lesion, for the circle of Willis is a common site for aneurysms causing a subarachnoid hæmorrhage. Partial aphasia or hemiplegia is not of use in true localization as a rule. The fact is that the signs and symptoms are strikingly intense as compared with the paucity of focalizing indications.

Ophthalmoscopic examination may reveal papillœdema and perhaps punctate hæmorrhages. Sometimes larger amounts of blood are seen under the retina and the lower part of the field may display a considerable subhyaloid hæmorrhage. This is said to be due to extravasation along the optic sheaths.

A massive albuminuria may be observed. In most of the reported cases the absence of renal disease has not been proved, but that is not of immediate importance, for the protein clears with the patient's recovery. This sign may be a source of diagnostic error, though it has often been noted in cases of cerebral disturbance. Claude Bernard, in describing his classic experiments, remarked: *"L'albuminurie peut donc passer dans les urines par suite d'un état particulier du système nerveux."*[2]

Glycosuria is also found in some cases, as in the instance related below. Acetonuria, equally important and probably equally common, but more clinically neglected in cases of cerebral disease, may also occur. Its importance where the question of diagnosis is concerned needs no labouring.

Herpes zoster has been observed during the recovery period and is caused by the local irritation of the posterior root ganglia by blood that has gained access to the neighbouring meningeal pocket.

Treatment.

Obviously there is only one thing to be done besides insisting upon absolute rest in bed, that is, lumbar puncture. But will the withdrawal of fluid tend to cause increased or recurring hæmorrhage? It seems doubtful if there is such a risk, except perhaps in the presence of the large aneurysm, and here the outlook is very grave, whatever is done. Probably the best answer to this question is given by Symonds.[2] He advises that lumbar drainage be used freely in the early stages while symptoms of dangerous compression are present. Later he counsels caution, and to avoid the danger of restarting the bleeding, suggests that if symptoms require further withdrawal of fluid, this should be done only with the patient recumbent, and that the manometric reading should not be reduced below 100 millimetres of fluid.

Prognosis.

In elderly patients, when it is known or assumed that the blood vessels are diseased, the outlook is grave; even though recovery takes place, the chance of recurrence is great. In younger subjects the outlook is better. It is possible that a leak may be permanently sealed, but there must be always an uneasy feeling that the accident will be repeated at any time. Is there any means of knowing how long the patient may remain untroubled by a similar visitation? Frankly, there is not. But months or even years may elapse between attacks of bleeding; even many years of immunity have been recorded.

It will be seen that it is important to recognize the slighter hæmorrhages in order that a guarded prognosis may be given and that warning may be issued against indulgence in those types of exertion likely to favour rupture of a weakened spot in the cerebral arterial system. A small leak may rapidly be followed by a severe and fatal leak, as in the

case reported by Reid and Gleave.[3] This patient felt faint and vomited, had low occipital headache and pain in the back, and felt feverish, but was able to walk some distance to consult her doctor. Shortly after this a rapidly fatal leak occurred. Many similar cases are reported.

Illustrative Cases.

CASE I.—A man, aged seventy-one years, suddenly fell unconscious while at work. There was some spasticity of one arm and leg. The pulse was 60 per minute, the systolic blood pressure was 110 and the diastolic pressure 60 millimetres of mercury. Lumbar puncture revealed bloody fluid. Urine contained acetone. Leucocytes numbered 20,000 per cubic millimetre. The patient died after six days with a rising temperature. At autopsy a ruptured sacculated aneurysm was found on the right middle cerebral artery.

CASE II.—A man, aged forty-seven years, suddenly collapsed and became comatose. Two months before he had suffered a "paralytic stroke" affecting the right arm and leg and speech, and recovery had not been entirely complete. He was curled up in bed, lying on the right side, was extremely irritable, and vomited. There was slight conjugate deviation of the head and eyes to the right. All reflexes were exaggerated. The systolic blood pressure was 178 and the diastolic pressure 115 millimetres of mercury, the pulse rate was 64 and the temperature 35·6° C. (96° F.). The urine contained a little albumin. Eighteen hours later he recognized friends, but he was confused and his memory was impaired. He was still curled up and very irritable. The systolic blood pressure was then 210 and the diastolic pressure 180 millimetres of mercury. The plantar reflexes were extensor. The neck was stiff and Kernig's sign was present. The cerebro-spinal fluid was under increased pressure and heavily blood stained. The patient died a week later in coma. *Post mortem* examination revealed a ruptured aneurysm of the middle cerebral artery. The serum had reacted to the Wassermann test, but whether the vascular change was due to syphilis or not was doubtful, there being little other evidence of endarterial disease.

CASE III.—A boy, aged about ten years, collapsed while paddling at the seaside and complained of severe headache. He rapidly became unconscious, and on admission to hospital a catheter specimen of urine contained sugar and ketones. On the strength of this evidence he was assumed to be a diabetic in coma, and insulin and glucose were administered. The abnormal constituents of the urine soon disappeared, but he remained in a stuporose condition. When seen at this stage, he was characteristically irritable, there was definite neck rigidity with head retraction, and Kernig's sign was present. There was a slight febrile reaction. The disks appeared slightly blurred. Lumbar puncture yielded brightly blood-stained fluid under increased pressure. He gradually improved and made a good recovery.

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THE ROTATION OF THE EYEBALL.

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IT seems to be the general opinion among ophthalmologists and modern anatomists that wheel-like rotation of the eyeball is impossible. As an expression of this opinion the statement of Mr. Herbert Fisher[1] may be quoted: "Rotation, as such, of the eyeball never takes place in health when all the extraocular muscles are working normally." Veterinary anatomists have apparently always held the opinion that the eyes of domestic animals can rotate. Indeed, the original name employed by veterinarians for the oblique muscles was *musculi circumagentes*, in consequence of their producing this wheel-like rotation of the globe of the eye.[2]

As a matter of fact, rotation of the eye is readily witnessed in those animals that possess slit pupils, and especially can this be seen in the tortoise. In animals with laterally placed eyes, nodding movements of the head obviously tend to produce rotation in the eyes.

It is to be noted that the double rotatory movement executed by the oblique muscles is altogether involuntary and that it is constantly produced when the animal inclines its head to one side: doubtless to maintain the visual axis always in identical relation with the same point of the retina. This movement is well seen in man when the head is brought round to either shoulder, and the eye then pivots in the orbit in an inverse direction to that to which the head inclines, so that a mark placed at the upper part of the iris when the head is straight would occupy the same position after the lateral movement. Simultaneous in both eyes, this pivoting is executed by certain muscles in each, the superior oblique for one, the inferior oblique for the other, according to the direction in which the head is turned.[3]

No statement could be clearer or more definite than this, and yet the fact is omitted in most modern text books of anatomy.

The older generation of human anatomists held the idea that the human eye could rotate; Cloquet,[4] for instance, states that the oblique muscles cause the eye to "experience a rotatory motion". But, so far as can be ascertained, anatomists have put forward no absolute proof of the rotation or measured the extent to which it can be carried.

In order to determine the degree of rotation of the human eye, the writer selected a series of individuals in whom some pigment spot on the iris permitted an estimation of the degree of movement. The apparatus consists of an optician's test frame fitted with a horizontal bar, to which an adjustable plumb line is attached. This is fitted to the subject and the plumb line adjusted to the pigment patch. A large protractor, marked off in 5° intervals, is placed behind the head and the mid-line of the head is marked by a chalk line down the forehead and face. The subject is told to look at a distant object and keep his gaze fixed on that point. He then moves his head slowly towards the shoulder. The pigment patch is then seen to keep behind the plumb line until a point is reached at which it remains steady and the plumb line leaves the patch. This point represents the maximum angle through which the eye has rotated, and the angle is read off on the protractor. It is almost constant in all individuals tested and in the whole series ranged from 5° to 7°. This method of observation is only approximate, since the rotation of the eye may lag behind the movement of the head.

Comparative anatomists and an older generation of human anatomists regarded the oblique muscles as having production of rotation of the globe as their main function. It appears from more recent accounts of the movements of the eye that the synergic action of the oblique muscles is in danger of being regarded as their principal function and their undoubted action as prime movers in causing rotation is being lost sight of.

The conjugate rotation of both eyes is obviously a reflex which is easily tired, since after repeated testing of one individual the rotation of the eye ceases temporarily and no pains on the part of the observer can make the eye rotate until the subject is rested.

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SUPERFLUOUS HAIR: ITS PERMANENT REMOVAL BY ELECTIVE ELECTROLYSIS ET CETERA.

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LITERATURE on the treatment for the permanent removal of hair (as met with in up-to-date text books upon dermatology) is by no means conclusive as to the best methods to be adopted in cases of hypertrichosis.

In *The Lancet* of May 16, 1931, some remarks were made upon this subject under the heading of "A Cosmetic Questionnaire"; these remarks arose from a paper appearing in the *Dermatologische Wochenschrift*, April 4, 1931. I obtained a copy of this article with some difficulty, but unfortunately it was stolen. It certainly suggested that some dermatologists had had good successes in some cases treated by X ray therapy alone. I also have had good results with X rays alone. However, as I have witnessed so many untoward after-results occurring in so many patients who have been treated by X rays for hirsuties and other troubles, I feel that I cannot accept the use of X rays as a routine method of treatment for the permanent removal of hair. I have seen the chin bound down to the neck by scar tissue, in which a plastic operation was necessary for the disability. The skin of both cheeks, neck and the upper part of the chest was one continuous mass of scar tissue. That, however, was a result of treatment by X rays administered for hypertrichosis some thirty years ago, before it was known that such trouble would occur. But quite recently I was consulted by a patient for a thickening of the skin of the face following upon two treatments with X rays; the patient stated that he was suffering agonizing pains in the thickened parts. He had been treated with X rays two years previously; only two treatments were given, and apparently the treatment was administered by one with an up-to-date knowledge of X ray therapy. And it appeared to me that idiosyncrasy could alone account for such a result. I have seen untoward after-results occur ten, fifteen and twenty years after the application of X rays. In some of these cases the unfortunate results have followed in patients who had received only the mildest exposures. Thus I feel that the use of X rays alone for the permanent removal of hair is, in my mind, open to question.

I have certainly obtained permanent removal of hair, both intentionally and accidentally, by X rays and radium, especially in the treatment of malignant disease. And I have seen these results obtained without being followed by any untoward after-results, at any rate up to the present. However, there is a percentage risk following X ray and radium treatment, applied sufficiently to cause permanent baldness. And if treatment in these cases is undertaken merely for cosmetic results, only such treatment should be applied as will assuredly not be followed by results which may be much worse than the disease.

Under the heading of untoward after-results may be mentioned: telangiectasis, atrophic spots, cicatrices, and even keloid, neuritis and peculiar irritating nerve sensations, like formication *et cetera*, leucodermic or melanotic disfigurements; sometimes, unfortunately, even malignant disease may follow.

In my method of treatment I aim at obtaining the beneficial effects of radium, X rays and electrolysis, separately or in combination, in accordance with the nature of the condition presented for treatment. The radio-sensitivity and position of the vital structures for hair growth must be considered.

In 1893 I read a paper at a meeting of the Victorian Medical Society describing cases of hypertrichosis treated by electrolysis, which was the recognized treatment for such cases at that time. I will now deal with some of these cases and describe the changed method of treatment which I adopt today.

CASE I.—A young man had a strong growth of dark hairs above the bridge of the nose and connecting eyebrows. As the patient complained of the surly expression and stern appearance it produced, his family wished him to have treatment. A suggestion of shaving the part was not acceptable to him; the hair was strong and growing slantwise, all the hairs pointing in one direction. The follicular hair shafts were from six millimetres upwards in length, and thus the application of the needle was difficult, and I probably many times pierced the hair follicles and destroyed the hair papillæ of adjoining hairs. However, the result of treatment was satisfactory. The hair was permanently removed, but there were some slight depressions, due to the large amount of electrolysis required to obtain destruction of the hair.

Elective Electrolysis.

When the hair papillæ are deeply situated, my custom now is to give the part a preparatory treatment with X rays, using a filter of 0·5 millimetre

of aluminium and such a strength that the hair could be easily epilated in about fifteen days' time.

This X ray exposure alters considerably, for a certain time, the tissues in connexion with the hair follicles. There is a lessening of the reaction of the cutaneous tissues to external exciting influences, such as pressure *et cetera*. Thus if a skin on pressure gives the triple response (Lewis), it will also give an exaggerated reaction to electrolysis. This condition can be modified by giving a suitable treatment with X rays beforehand. And the condition of the skin then allows of a more definite application of the point of the needle to the hair papilla, due to the comparative dryness of the follicular structure, which favours an elective electrolytic action to the deeper structures.

By this means one obtains a much more certain destruction of the hair, treatments for recurring hairs are lessened accordingly, and the tell-tale pimples of electrolysis are likewise much less apparent.

Another patient mentioned in my paper was a man with double eyebrows. I merely mention this case now as evidencing the futility of trying to get rid of hairs by extracting them.

The patient, when three years of age, had probably been painted on the forehead with a nitrate of silver lotion which had darkened the lanugo hairs, making them quite conspicuous. An over-anxious mother and nurse, alarmed at this apparent growth of hair, pulled them out and kept on extracting them, and when the boy was old enough, he took up this method of hiding his deformity, the hair evidently increasing in strength due to this process of epilation. For twenty-five years the hair was epilated and then I removed the growth by electrolysis. But, as in the previous patient, a judicious application of X rays would have lessened the number of treatments required for the permanent removal of the growth.

In cases like these, in which the hair shafts are long, I use silver or platinum wires in place of needles; the wire is sharpened to a point, the actual point is cut off, in order to lessen the risk of the wire on insertion piercing the wall of the follicle and so missing the direction of the hair papilla. The direction of the follicles varies, but the wire, thus prepared, is very pliable, and when passed into the follicle, travels down by the side of the hair shaft. Judgement must be used not to pass the wire too far and so to miss the hair papilla. One or two hairs should be extracted in order to get an idea of the lengths of the hair shafts, which vary considerably, even with similar looking hairs. I generally use three wires, so that no time is lost in transferring my attention from one follicle to another. The three wires are connected with the main wire coming from the negative pole of the battery. When wires are used in this way, the electrolytic action is not so apparent as with needles used in the ordinary way, and so judgement as to the time of application must depend to a certain extent upon experience. However, the time of application is much the same as with ordinary electrolysis, but the pain is considerably less, owing to the altered state of the cutaneous tissues following upon the X ray treatment.

In many elderly women long, coarse hairs develop upon the chin. They do not like the idea of shaving or using depilatories, and wish to have the hair permanently removed. This elective electrolysis process is very successful in these cases.

In my paper I mentioned several cases of ladies' moustaches. In these patients the growth of hair upon the upper lip varies tremendously, from a genuine moustache down to one of imagination, and so no one form of treatment is suitable for all cases. A very common condition is a few dark hairs, forming more or less a tuft of hair at the outer borders of the upper lip, and there may be some small hairs generally upon the upper lip. An examination shows that hair shafts of the noticeable hairs are only two or three millimetres in length. I have found in many of these patients that if the noticeable tufts at the outer borders of the lip are removed, the patient is satisfied with a decolourizing application for the small hairs, or the use of abrasive measures, which certainly at times produce permanent removal of hair. For these cases I had radium needles made so that I could get the elective action of the hard β rays to act upon the hair papillæ of the part affected. These needles contain an equivalent of five milligrammes of radium element. They are made of Monel metal and the thickness of the wall of the needle is 0·2 millimetre.

The manufacturer in Belgium warned me that the production of these needles was at my own risk, as they would not stand rough usage. The standard radium needles of today have a thickness of 0·5 millimetre of platinum and so can stand a considerable amount of pressure by forceps *et cetera*, but, of course, practically no β radiations can be applied when these needles are used. However, these Monel metal needles have stood ten years' constant usage without any apparent damage up to the present.

Monel is a natural alloy metal of copper and nickel, is rust resistant and non-corrosive; its density is 8·8, that of nickel being 8·28. And as 0·13 millimetre of nickel cuts off only 50% of the β rays and it takes 1·1 millimetres of that metal to cut off 99% of the β rays, a good deal of β radiation will therefore pass through needles with a wall thickness of 0·2 millimetre of Monel. These needles, however, are invaluable for cross-fire radium therapy of rodent ulcers *et cetera*. The radium preparation is contained in a channel of the shaft of the needle, 12 millimetres in length, the exact position of the channel being marked on the needles and its presence can be proved by its action upon a photographic plate. It is probably correctly stated that 8·5 millimetres of soft tissues cut off 99% of β radiations, and that one millimetre will cut off 50%. Viol, a physicist, according to Newland Simpson's book, states that seven-eighths of β radiations are cut off by four millimetres of soft tissues, and Failla, a physicist (MacKee's book), states that seven-eighths of the β radiations are cut off by three millimetres of soft tissues.

It is strange to find scientists differing so widely in an important matter like this.

However, I have found that the very hard and highly penetrating β rays are of considerable therapeutic value—when used for the part they play—in what I have termed collision value.

Collision Value.

At a medical congress held in Brisbane in 1920, I read a paper upon the value of cross-fire radium therapy in the treatment of cancer and other conditions. I used the term collision value as if visualizing X rays from one direction meeting or coming in contact with β rays coming from the opposite direction. I said that I could not speak from a scientific point of view of what really occurred. However, I mentioned several experiments which I had carried out (electronization on the metamorphosis of insect life) which showed the enhanced value of the treatment of radium when carried out in this way. One of my experiments was the exposure of three weeks old larvæ of the *Bombyx mori* to three hours' treatment by radium, with mica intervention only at five millimetres distance; during the last ten minutes of this treatment a cross-fire with X rays was given.

Similar experiments were carried out with the same dosage, except that X rays were applied a little while after the radium treatment had finished. In these experiments the subsequent effects were much greater in the cross-fire treatment, and suggested to my mind a collision value by X rays (electro-magnetic waves) coming into contact with the electron or β particles.

Recently reading a book by Professor Eddington, I found that he made statements which show that my ideas of collision value, founded upon experimental and therapeutic results, are probably correct.

Professor Compton, of Chicago, has studied what happens when X ray radiation falls on electrons. He finds that "the radiation is scattered really as though it consisted of material particles of light or photons moving as separate detached units, thus like bullets on a battlefield, and hitting all electrons which stand in their way". A veritable battlefield certainly supports my collision value. However, when one reads Jeans's book, "Mysterious Universe", one meets with the supposition that "electrons and protons, the fundamental units of which all matter is composed, can appear, now as particles and now as waves, and that radiations of all kinds can appear similarly as particles and as waves". It would appear that the difference in radiations depends upon the velocity of the quanta or packets of waves of which radiations are formed. And so, in order to destroy these tufts of dark hair of the upper lip previously remarked upon, I aim at producing a veritable battlefield in and about the hair papillæ and the vascular supply to their hair follicles *et cetera*, with as little probability as possible of incurring any untoward after-results of radium treatment in the future. Generally two Monel needles, as previously described, are covered with rubber tubing, 2·5 millimetres in thickness, and applied externally. The rubber tubing prevents the effects of the characteristic rays from the Monel metal, and, by increasing the distance, allows of a wider effect upon the parts to be treated. Under these conditions the quantity of β rays is cut down considerably, but many of the hardest or most penetrating will radiate into the parts where treatment is more particularly desired. Specimens of radium giving γ rays are then applied internally, the application pressing upon the mucous membrane immediately opposite to the external applicators. The time of application or dosage will depend upon the strength of the applications and the thickness of the tissues under treatment; but generally about one and a half hours' application can be arranged for. The hair should fall out in about three to four weeks' time. If further similar applications are required, I apply the β ray applicators from the inside. If there are still a few recurrent hairs, it is advisable to remove them by elective electrolysis, but only strong hairs should be so removed, as a few fine hairs help to prevent the part appearing too bald in comparison with the surrounding parts. In this application one aims at obtaining collision value in those tissues of the skin most vital for the growth of the hair. There is no doubt that the introduction of standard radium needles, applied for a long time, has greatly improved the efficiency of radium treatment in malignant growths. This is in all probability largely due to the fact that cross-fire therapy is more efficiently applied by numerous needles than it was when tubes, although containing more radium, were fewer in number and were placed much further apart. Five one milligramme standard needles suitably inserted into a growth will have much more therapeutic effect than is obtained when one standard needle is inserted, containing five milligrammes of radium. Acetate of thallium, applied as an ointment, has given good results in some cases of hirsuties, but the risk of untoward after-results (including intractable ulceration of the skin) is well recognized, and I have not been using this method for some years. Abrasive measures are undoubtedly sometimes followed by permanent loss of hair, but such treatments are as a rule unsatisfactory, and depilatories are generally objected to by patients.

Ingrowing Hair.

Occasionally one meets with ingrowing hair, generally on the face. The patient describes how he cuts himself, owing to the razor blade catching in the hair. The hair may be recognized growing laterally in the epidermis. Sometimes this dark line indicating the shaft of the hair is about one centimetre in length. I have had good results with X rays in these cases. A depilating dose of X rays is administered and the hair falls out. When the new hair forms and grows forward, it appears to have lost the tendency to grow laterally into the epidermis and comes out of the mouth of the follicle in a natural way. If a few hairs acquire the old habit, they should be epilated or treated by electrolysis, when a good result often follows and so renders further application of X rays unnecessary.

Pathological Hair.

Occasionally (more noticed in women) hair takes on an unusual growth, which may be very inconvenient and unsightly. The following case illustrates the great value of X ray treatment in one of these patients.

A young woman, just qualified at the university, complained of the hair of her legs having developed during the last year of her studies into long dark hair. It was just at the time when short skirts and light stockings had come into fashion. The hair was so long that it penetrated the stockings, and, as she remarked, looked like spikes. She was to be married in about four weeks' time and wished me to get rid of her trouble by depilating the parts affected with X ray treatment. She refused my advice to shave or use depilatories. I then administered a depilating dose of X rays. She wrote to me some time afterwards and told me the X ray treatment had been a complete success, and, as a matter of fact, the hair that returned was quite normal in appearance.

My experience is that pathological or functional hypertrichosis (the latter occurring on girls' faces at about the age of fifteen or sixteen) generally subsides as the unusual stress or functional activities causing these conditions discontinue.

The effect of nerve shock upon the growth and destruction of hair are well known. However, I wish to put upon record two rather extraordinary cases which have come under my personal experience.

The first of these cases is one of permanent loss of hair due to shock.

A lady, carrying her twins, one under each arm, tripped and fell when seven steps from the bottom of the staircase. She said she received a great shock and she does not remember whether she put her arms out to save her face or not. There was a great "hullabaloo" when she reached the floor, but all were picked up by the rest of the family and all were apparently quite well in a few minutes' time. However, in three or four weeks' time the hair of her head started to fall out in smooth round patches (*alopecia areata*) and, in a few weeks' time, her body was completely denuded of hair with the exception of her eyelashes. Her hair has never returned, and it is now over thirty years since the accident. She also developed a very intractable *lichen planus*, a skin trouble usually considered to be of neurotic origin. This condition continued until she again became *enceinte*, when it cleared up permanently.

Still we cannot expect to use shock as a means of producing permanent removal of hair.

The second case is one of sudden blanching of hair. This case also shows the effect of shock upon the growth of hair.

The patient stated she was benighted. The night was very cold and foggy; she was terribly frightened. Her husband and friends were out searching for her all night. In the early morning some of the party, including her husband, found her. Her husband came running up to her in his excitement, but stopped short just as he reached her. She said to him: "What is the matter?". He replied: "Your hair has turned quite white."

I had every reason to believe the history in this case to be quite true, although it is very difficult to understand, as Darier states in his text book, 1920, at page 417:

I do not know of any observation of sudden canities occurring in the course of the last four years, which have certainly been prolific of unspeakable horrors.

Darier was referring to the Great War, and it is certainly strange if no case occurred during that time.

Untoward After-Effects.

Of the untoward after-effects previously mentioned as occurring in patients who have been treated with radium, I will mention three of the commoner conditions and describe the treatment which I have introduced for these conditions with good results.

Leucodermic Atrophic Spots.

The white spot is noticeable at a few feet distance, but, on closer examination, one generally finds there is a certain amount of *atrophia cutis*.

For these conditions I arrange to deposit a certain amount of metallic substance in the area (copper, gold or steel) in order to get an appearance resembling the surrounding skin. On my own left wrist I have practised the deposit of metal in the skin in order to get the colour of deposit suitable for the case in question. The ordinary apparatus for electrolysis is used, but the needle is attached to the positive pole, so that no electrolytic action takes place, but a dissolving of the point of the needle occurs and a deposit is made in the skin. A good deal of the deposit fairly quickly disappears, so that a coloration is produced, which appears to the patient at first as too much colour. The point of the needle, which is attached to the positive pole of the battery, is inserted into the skin for about two millimetres or more, in accordance with the nature of the skin affected. The punctures of the needle are applied about two millimetres apart, in the form of squares, and then one puncture is made in the centre of the square. I generally find about one milliampère gives sufficient current for this purpose. On the face, where these disfiguring lesions occur, the skin is loose and can be held tightly between the fingers and thumb of the left hand. This steadies the part for the operation and helps to lessen the sensation of the needle whilst the current is applied. If the patient is very sensitive to the treatment, a local anæsthetic is necessary.

Telangiectasis.

It is tempting to pass the point of a needle along the length of the disfiguring vessel and blanch it by electrolytic action; but the condition frequently recurs after such treatment and similarly recurs after freezing with carbon dioxide. However, I puncture the vessels with the electrolytic needle (the needle to be inserted for two or three millimetres, and punctures to be made two millimetres apart, a current of one milliampère being used). The part is then painted with celloidin, which, if properly made, should stay on for two days. I find that this method is fairly successful in destroying telangiectases (these appear to be made of capillary loops, which accounts for the punctures being more successful than a linear insertion of the needle).

Formication.

Perverted sensation (formication) of the skin after radium treatment gives some patients considerable annoyance.

I use the same treatment for these cases as I have found successful in treating certain cases of recurrent *herpes facialis*.

Radium disks, which contain radium covered with mica and mackintosh only and so giving β radiations, are applied over eight layers of lint. The spread of the radiations of each disk is equal to the size of a halfpenny, and so a considerable area can be treated at a time if six of these disks are used. Two hour treatments are given, with two week intervals. I have found that about six or eight treatments will generally clear up permanently a recurring *herpes facialis* of children. A similar but less extensive treatment is successful in the treatment of the perverted sensations.

NOTE UPON DIASTOLIC PRESSURE OF ZERO.

By PAUL G. DANE, M.D. (Melbourne),
Melbourne.

FOR some twenty-five years there has been a considerable amount of discussion upon the subject of blood pressure.

I have read whatever has appeared upon the subject in the ordinary clinical journals, but I do not recollect ever having noticed any remarks upon a diastolic pressure reading of zero, except many years ago, I think in *The Journal of the American Medical Association.*

Some fifteen years ago I showed to some students a patient suffering from syphilitic aortitis with a high systolic pressure and a diastolic pressure zero.

When I informed some of my colleagues that I had seen a patient with a diastolic pressure that was not measurable on the clinical syphomanometer, a smile of incredulity was all I received.

However, I have since seen three more patients with a diastolic pressure of zero. The last was a young man of thirty-two years, who had apparently complete aortic incompetence, the ætiology of which was undetermined. There had been no syphilis and no known rheumatic infection, but there had been three attacks of pneumonia.

Patients with a diastolic pressure of zero show marked aortic pulsation, and a loud, sharp, snapping sound is heard on auscultation of any of the large arteries. In one of my patients this loud, sharp sound could be heard in the erect position by merely approaching one of his limbs, without actually making contact with his body by the ear or stethoscope.

It appears that the sound which is held to indicate the reading of the diastolic pressure is caused by the "snap" of the sudden distension of the artery by the pulse wave. This explains why it is sometimes so difficult to estimate the diastolic pressure in subjects with arterial thickening or with myocardial weakness, or both together.

It is always wise to estimate the blood pressure with the patient in the erect position as well as in the recumbent. We pass the larger portion of our time erect, and most of us work erect or semi-erect. In aortic incompetence the pulse pressure will be found higher in the former than in the latter position, whereas in cases of high blood pressure the reverse is generally the case, that is, the diastolic pressure is higher and systolic pressure lower in the erect than in the recumbent position.

The prognosis appears to be very grave in patients with a diastolic pressure of zero. Two of my four patients died very shortly after being seen, and it was not the treatment that killed them, for I gave them none; but the last patient appeared to have a fair reserve of cardiac power, in spite of his defective circulation.

It would be interesting to know how the brain "carries on" under such conditions. Can it function with arteries that are full to repletion and empty again seventy times each minute, or is there really some blood pressure in the arteries notwithstanding that the instrument reads "zero", or is there some mechanism in the cerebral arteries that gives a more constant pressure of blood to the brain?

The opinion and experience of others more experienced in cardiac disorders would be appreciated upon this subject.

It must be clearly understood that the loud snapping sound heard in subjects with a diastolic pressure of zero can be heard apart from using the blood pressure estimating instrument; it is heard without any pressure being put upon the artery whatsoever.

Reports of Cases.

FOUR CASES OF SNAKE BITE.

By C. HUMPHREY LLOYD, M.B., Ch.M. (Sydney),
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THE four cases of snake bite presented in this report have a practical value. The first case was fatal; the second, third and fourth patients recovered. The first case was hopeless, I think, as no effectual early treatment was carried out, and the treatment given by me was too late to do any good. The second and third patients were bitten through their trousers, and this may have prevented any venom from being injected. In the second case the snake bite was doubtful, as the patient did not see the snake and he was unaffected. In the third case the patient saw the snake and said it was a tiger snake; his being bitten on the thigh while working would make this feasible. As he was so unaffected, it is doubtful whether he was injected with venom. If he was injected with venom, his preliminary treatment may have done some good. This is unlikely, however, as preliminary treatment was given so long after the bite. The same may be said of the second case. In the fourth case the patient was bitten; the snake was seen to bite his finger and it was definitely a tiger snake. In this case the early treatment, the administration of antivenin and treatment in hospital, I think, saved the patient's life.

Case I.

On October 14, 1931, R.M., a male, aged two years and five months, while playing, was bitten by a death adder on

the left leg above the ankle, on the outer side. The time that elapsed from bite to arrival at the surgery was about twenty minutes. The leg had been washed with potassium permanganate solution, crystals of potassium permanganate had been rubbed on, and a loose tourniquet had been put on the leg below the knee. The leg had not been incised. This treatment was, of course, useless. The patient appeared to be in moderately good condition, although he was pale and had a somewhat anxious and drawn expression. The pulse was of good volume, its rate being 96 per minute. A tourniquet was applied round the thigh. The wound area, where four punctures could be seen, was incised and a vein in the leg was opened and some ounces of blood were taken off. He was taken to hospital. The tourniquet was loosened for from half to one minute every half hour with no immediate symptoms. The pulse rate increased, though still of good volume, the general condition of the patient was moderately good, but he became more anxious and restless and paler. Hypodermic injection of strychnine sulphate 0·32 milligramme (one two-hundredth of a grain) was given, and the leg bled again without much blood escaping. About two hours after being bitten and after the tourniquet was relaxed for the third time, the patient's pulse suddenly faded out, and with one or two sudden convulsive movements of the body, and the hands clutching at the throat, death supervened. Artificial respiration and other stimulants were of no avail.

Case II.

On December 17, 1931, D.M., a male, aged twenty years, a farm hand, was bitten by a snake (said to be a tiger snake) on the right thigh while lying in some grass at the creek side getting a drink from the creek. A tourniquet self-applied was ineffectual. About ten minutes after the bite an effective tourniquet was applied and the wound was incised previous to admission to hospital. I was attending an accident some miles out of town in the bush when the patient was brought in, and the matron and staff treated the patient after telephoning Dr. Burton at Texas, thirty-five miles away. The treatment, which was successfully given, comprised rubbing in crystals of potassium permanganate, hypodermic injection of strychnine sulphate 1·0 milligramme (one-sixtieth of a grain), and intravenous injection of 1,500 units of tiger snake antivenin (Commonwealth). The patient complained of a feeling of nausea on admission. His temperature was 37·2° C. (99° F.) and pulse rate 68 per minute. This patient had no further symptoms beyond stiffness in the leg and some pain next day, and was discharged on December 22, 1931, quite well.

Case III.

On January 13, 1932, E.R., a male, aged thirty-two years, a worker in tobacco growing, was bitten, while working, on the left thigh by a snake, said to be a tiger snake. A tourniquet (effectual) was applied and the wound was incised and sucked previous to admission to hospital some ten minutes after the bite. The patient ran some distance to his house, where he was treated. He was rushed to hospital and 1,500 units of tiger snake antivenin were given intravenously and a vein in the leg was opened and a small quantity of blood let. Hypodermic injection of strychnine sulphate 2·2 milligrammes (one-thirtieth of a grain) was given. This patient's condition was never bad, his pulse rate was 68 per minute and of good volume. He had no symptoms beyond pain from the tourniquet and was discharged three days later, quite well.

Case IV.

On March 5, 1932, P.N., a male, aged twenty-seven years, was brought to the surgery, having been bitten on the index finger of the right hand by a tiger snake, which was seen a few feet away from a baby by its father, who with P.N., tried to kill it. P.N. chopped at the snake with a tomahawk and pinned it down, the snake striking back and fastening on to his finger. This snake was afterwards killed and was a tiger snake about five feet long. A tourniquet which was tight enough to do some good, was applied immediately around the first phalanx and the wound was incised and sucked. About ten minutes later an effective tourniquet was applied round the upper arm. (He was also given rum enough to make him very drunk.) At hospital an hour later he was given 3,000 units of tiger snake antivenin intravenously and the tourniquet was loosened for half a minute a few minutes after this injection. Within a few minutes his pulse became very weak, the patient called out that he was gone, and muscular twitchings developed in arms and body. Hypodermic injections of strychnine sulphate 2·2 milligrammes (one-thirtieth of a grain) and adrenaline (1 in 1,000) 0·6 mil (ten minims) were given, when the pulse became good again, its rate being about 68 per minute. About an hour later the pulse again became weak and hypodermic injections of strychnine sulphate 1·0 milligramme (one-sixtieth of a grain) and adrenaline (1 in 1,000) 0·3 mil (five minims) were given. Again the pulse and general condition improved. The median and radial veins were opened and about 450 cubic centimetres (fifteen ounces) of blood were let. The patient seemed better after this and did not get symptoms on loosening the tourniquet. About an hour later the pulse again became weak and the rate was 108 per minute. A hypodermic injection of strychnine sulphate 2·2 milligrammes (one-thirtieth of a grain) was given with a good reaction. About three hours after the first injection of antivenin a further 1,500 units were given intramuscularly. The tourniquet was loosened more often and for a longer period, and the patient had no more symptoms and settled down into a good sleep with a strong pulse, its rate being 68 per minute. He vomited all the rum about 12 midnight. The patient complained of pain in the arm and was restless at about 3 a.m. He was given a powder of aspirin, phenacetin and caffeine citrate and was fairly comfortable for the rest of the night. For the next few days he complained of his arm being stiff and painful. His hand and finger were swollen. This subsided by March 10. On March 11 he developed a profuse urticaria on the body, which cleared up by March 14, and beyond some stiffness and loss of power in his right hand, he has quite recovered.[1]

"SODIUM AMYTAL" IN OPHTHALMIC SURGERY: USE OF THE "BRIDLE SUTURE".

By CLIFFORD COLVIN, M.B., Ch.M., D.O.M.S.,
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FOLLOWING Dr. Blakemore's instructive article on the use of "Sodium Amytal" in ophthalmic operations, I should like to report a case in which use was made of the anæsthetic dose.

The patient, a female, E.G., aged about sixty years, is an inmate of the Orange Mental Hospital. She is suffering from secondary dementia, is restless and mentally confused, and would not have remained quiet after ether anæsthesia; she is not intelligent enough to be able to cooperate. She had double senile cataracts and was unable to feed herself, so the question of their removal was raised by Dr. Hilliard, the Medical Superintendent.

"Sodium Amytal" is extensively used here for surgical operations, and it was decided to remove the right lens under that anæsthetic. Dr. J. Rossell kindly undertook to administer it. The usual procedure of splitting the dose into portions was followed, and a total of 0·9 milligramme (fifteen grains) was given. The body weight was about 60·5 kilograms (nine and a half stone).

The patient was brought to the theatre deeply narcotized, but when her lids were opened, she showed some resentment. "Codrenin" was then used as a local anæsthetic, but when the eye was touched, resentment was again manifested. A few drops of ether were then used (not more than half an ounce) and the operation was commenced.

After the insertion of the speculum, the first step was the insertion of a strong suture on a curved needle round the tendon of the superior rectus muscle. This

[1] This patient was seen on September 2, 1932; he had quite recovered the power in the right arm and hand.

was not tied, but was left long and held by the assistant. By gentle traction on this suture complete control of the eye is obtained, and there is no danger of the eye turning up. This is the so-called "bridle suture" as used in Viennese clinics.

The operation then proceeded in the usual manner, and not the slightest difficulty was experienced, the assistant having complete control of the movements of the eye.

The patient remained perfectly quiet for the next twelve hours, when a further dose of "Sodium Amytal" (0·18 gramme or three grains) was given to insure her remaining quiet. She was quiet for the next twenty-four hours; no vomiting or restlessness occurred.

The healing was uneventful and the result very satisfactory. Incidentally I might mention that recuperative power seems to be greater in mental patients than in normal patients with ocular lesions. The operation would have been very difficult or almost impossible (as Dr. Blakemore reports) had it not been for the use of the "bridle suture". This suture is very useful in any intraocular operation; its use is essential under general anæsthesia, but it can be dispensed with when the patient is intelligent enough to cooperate.

SOME OBSERVATIONS ON THE TREATMENT OF DEMENTIA PRÆCOX WITH "THEELIN".

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AND

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WHENEVER possible, treatment should, of course, always be based on ætiology and pathology, but in the presence of an urgent and devastating disease like *dementia præcox* treatment cannot wait for pathology, and any and every method of treatment which seems to offer a reasonable chance of success should, we think, be tried empirically and the results, whether successful or not, should be published.

The possibility that *dementia præcox* may be due to a profound disorder of metabolism caused by a complicated endocrine disturbance in which the ovaries and testes are specially involved is suggested by the following facts: (i) The onset of the malady during adolescence. (ii) The frequent occurrence of hypoplasia of the genital organs. (iii) The histological researches of Mott and his co-workers, who found in a large proportion of male patients a complete arrest of spermatogenesis and in all of them a greater or less degree of regressive atrophy in the testes, and in female patients analogous changes in the ovaries. (iv) the unhealthy sexual behaviour of most precocious dements, even in the pre-psychotic stage, resulting from a feeling of sexual inadequacy.

With this theoretical foundation we decided to try the effect on precocious dements of the new ovarian follicular hormone, "Theelin", discovered by Professor E. A. Doisy and manufactured by Messrs. Parke, Davis and Company.

We selected for treatment four young female patients with well developed symptoms, in whom the diagnosis was not in doubt.

Case I.

The patient is aged twenty-four years, and married, with two children, the younger being fifteen months old. There is a history of worry about her husband's unemployment and of mental conflict in connexion with having changed her religion when she married. Her present illness began with sleeplessness six months ago, shortly after the extraction of a number of teeth. She showed at first mental depression, loss of interest and loss of energy, and later developed auditory hallucinations and delusions.

When treatment with "Theelin" was commenced she was slow of thought, listless and indifferent, showed wrinkling of the forehead and suffered from auditory hallucinations. Her periods were regular, but she suffered from menorrhagia. She was given thirty-six daily injections of fifty units of "Theelin".

She shows a good deal of improvement, is brighter and more responsive, takes more interest in her surroundings and more care of her personal appearance, and works in the ward kitchen. Her physical health has improved, and she does not appear to suffer so much at her menstrual periods.

The amount of improvement is unfortunately not greater than that which could have been obtained by encouragement and occupation therapy alone, and we do not feel justified in attributing her improvement to the effect of "Theelin".

Case II.

The patient, aged thirty-five, single, has been ill for two years. She is unoccupied, restless and agitated. She is evasive in her replies to questions and talks past the subject. She often verbigerates. She is negativistic and frequently causes trouble by refusing her food. She suffers from auditory hallucinations. Her periods are irregular.

She was given thirty-six daily injections of fifty units of "Theelin", but does not show any improvement. There has been no return of menstruation.

Case III.

The patient is aged nineteen, and single. Her father was a Russian doctor who died during the revolution, and she herself was a girl of more than average ability. Her mental illness dates back four years. She is inaccessible, wanders aimlessly about the airing court all day, scribbles on the veranda and destroys her clothes. She laughs and talks to herself and verbigerates when excited. She is faulty in her habits, and at times is impulsively violent. Her menstruation is irregular. She received fifty units of "Theelin" daily for twenty-six days, but showed no change at all.

Case IV.

The patient is aged twenty-nine, and single. She was formerly a clerk and of bright and active disposition. The duration of her present illness is two and a half years. She sits about all day in constrained attitudes. She suffers from auditory hallucinations and often smiles to herself without apparent cause. At times she tears up her clothes. She is faulty in her habits. She suffers from amenorrhœa.

She was given twenty-six daily injections of fifty units of "Theelin", but showed no improvement.

Noting these unfavourable results, we thought that very likely the dosage of "Theelin" had been inadequate, so we decided to give more intensive treatment to a fifth patient.

Case V.

The patient is aged eighteen, and single. The duration of her illness is three months. She is dull, listless and unoccupied. She is slow in all her movements and has to be urged to take her food. She is faulty in her habits and makes sores on her skin by picking herself.

She was given an eighteen-day course of injections of "Theelin", starting with one hundred units and rapidly rising to five hundred units per day. The "Theelin" was not pushed beyond five hundred units per day, partly because of its apparent lack of effect and partly on account of the serious expense. The patient shows an appreciable improvement, both bodily and mentally, but no more than might have been expected as the result of the favourable conditions and the amount of individual attention given to her. There has been no return of menstruation.

It is recognized that the total number of patients treated with "Theelin", five, is very small. The question of dosage too is very important, and it may be that the amounts given were too small. The "Theelin" did not appear to have any bad effects, and the injections did not cause any pain or local reaction. The injections were all either subcutaneous or intramuscular.

To sum up, our experience, as far as it goes, tends to show that "Theelin" given in ordinary doses has no beneficial effect in cases of *dementia præcox*, and this finding is in harmony with the fact that castration or total deprivation of ovarian hormone does not produce *dementia præcox*.

Reviews.

CONVENTIONAL ENGLISH MORALITY.

MRS. JANET CHANCE has written a book, "The Cost of English Morals", which is likely to give rise to much controversy.[1] Sir Thomas Horder, in an introduction, describes it as a "brave little book", and he adds that its success is assured by the earnestness with which it is written and by the tremendous importance of its theme. The author opens her first chapter by stating that "we are paying too high a price for our English morals. They hamper intellectual life. And they debase sexual life." She sets out in her book to analyse English morals in their effect upon sexual life, to show that they cause much suffering and to ask whether they are worth the price that is paid. She points out that some of the accepted standards are good and essential to any wise code of sex morality. "It is good, for instance, that there should be insistence on some considerable measure of self-discipline, self-management and self-control in sex life; and that there should be emphasis on consideration for others as a large part of sex morality; and that there should be an appreciation of the frequent antagonism of the merely physical to the mental and emotional aspects of sex activity." She goes on to state that, though these standards are valuable, there are others that are far from sound and for which we are paying too heavily. She enumerates them as follows:

1. That sex is on the whole an unpleasant, if not a somewhat indecent part of life.
2. That knowledge of sex matters should be as restricted as possible for both adults and children.
3. That sex life should never be an end in itself.
4. That extra-marital sex life is in itself wrong, and jealousy on its account is justified in the married.
5. That purity is a finer ideal than vitality, and that there is a state called spiritual, which is a good state, and consists in rising superior to the body.
6. That there is no vulgarity nor cruelty in exposing the sex life of unmarried persons.
7. That the best interests of marriage are served by legal restrictions aimed at maintaining it, whether good or bad.
8. That there is too much sex interest and sex activity in modern life.
9. That in the double standard in sex morals the man's is the lower of the two.
10. That it is legitimate in the name of religion to impose standards of sex morality on those who consider the religion an illusion and the morality an evil.

The author accepts the opposite principles as more likely to result in health and happiness and sets them out as follows:

1. That sex life can be either pleasant and inspiring or unpleasant or degrading, and that it is for us to achieve the former and avoid the latter.
2. That if minds are supposed to be too weak to stand sex knowledge, the remedy lies in exchanging the enfeebling and confusing unrealities of orthodox education for an early acquaintance with the world as it is, and this in every sphere.
3. That sexual happiness apart from procreation is a good thing, and marriage and all other sex unions should be judged by their success or failure in achieving it.
4. That jealousy is a weakness to be corrected by understanding, sympathy and self-discipline. Adultery and the frigidity which is often its cause are both to be judged by the health and happiness they create or destroy.
5. That, in adults, the vitality arising from healthy sex experience is preferable to the purity of inexperience.
6. That present social behaviour in regard to extra-marital sex relations is uncivilized.
7. That legal interference with sex life should be limited to the care of the health of all concerned and to the safeguarding of the interests of the children affected, leaving the intricate question of the desirability of the union to the decision of the partners themselves.
8. That there is too little fine sex life in England.
9. That in so far as there is a double standard, men, though sometimes at great cost to others, are on the whole seeking a positive good; women are too often obstructive and negative.
10. That no religion should be permitted to use compulsion in furthering its aims.

Mrs. Chance throws down the gauntlet and states her case with obvious earnestness and conviction. Not many people will accept all the "opposite" principles enunciated by her. The point on which emphasis should be laid is that if there were more self-discipline, self-management and self-control, many of the problems which she discusses would not arise. At the same time there can be no question that a great deal of cruelty is inflicted on many people in the name of conventional morality. Moreover, those who inflict this type of cruelty are often what Mrs. Chance chooses to call the spinster-minded (we like the term). We do not propose to discuss all the points raised in this book. Rather would we, as Sir Thomas Horder suggests, leave each reader to determine how far the author is right in her contentions.

Of special interest to medical practitioners are the author's plea for adequate instruction on sex matters and her challenge to the medical profession to break its silence on them. She quotes from books, prescribed for use in schools, passages which show how inadequate instruction on sex can be in the teaching of physiology. Sex instruction should, of course, be given to children in their homes, and it is our impression that the "conspiracy of silence" does not exist to such an extent as it did a decade or two ago. Sir Thomas Horder replies to the author's challenge to the medical profession and reminds her that "the doctor is not a publicist; his whole tradition and training tend in the opposite direction and most of us think that this is all the better for the patient". He regards government health services as not being so restricted in action as the private practitioner, but he adds that no constructive legislation must be expected of a government until it is urged thereto by public opinion. Most people will agree with him.

[1] "The Cost of English Morals", by Janet Chance, with an Introduction by Sir Thomas Horder; 1931. London: Noel Douglas. Crown 8vo., pp. 127. Price: 5s. net.

CLINICAL PSYCHIATRY.

STUDENTS and practitioners who are interested in the problems of psychological medicine and who are seeking an easily read text book, will welcome Dr. Yellowlees's course of lectures.[1]

In his introductory lecture, the author deplores the lack of psychiatric teaching in medical schools and universities, and points out that the medical graduate is liable to commence practice with an inadequate knowledge of how to approach, or even to recognize, a problem in psychiatry. He admits that a cogent reason for a lack of interest in this department of medicine is the fact that text books in psychiatry are, in the main, written for the specialist, and that the mass of controversial theories found in these

[1] "Clinical Lectures on Psychological Medicine", by H. Yellowlees, O.B.E., M.D., F.R.F.P.S., F.R.C.P., M.R.C.P., D.P.M.; 1932. London: J. and A. Churchill. Demy 8vo., pp. 316. Price: 12s. 6d. net.

manuals deters the student from reading the fundamental groundwork. The lectures in this book are designed to present the clinical aspect of mental disorders.

It is pointed out that the student finds difficulty in discovering the parallel in psychiatry to palpation, percussion and auscultation in ordinary medical practice. Fundamental information as to the patient's mental state can be gauged only by observation of the patient's demeanour, and of the manner in which questions are answered.

Text books often fail to give any indication as to how a suspected psychotic patient should be questioned. The author admits the difficulty in laying down any specific rules, but useful and common sense information is given without the interminable list of questions that are tabulated in some text books.

Nine lectures are devoted to psychotic manifestations. There is no attempt made at any classification of mental disorders, but the various abnormal mental states are described and discussed, the reader being given an excellent clinical picture of each state in simple terminology. It is obvious that patients were shown during the delivery of the lectures, but the author has skilfully recapitulated them in such a way that the published lectures do not lose by the cases not being described in detail.

As each mental state is described, mechanisms underlying the production of symptoms are discussed and the general management is indicated in a practical manner. Useful guidance concerning the selection and administration of sedative drugs is given.

Criticism might be offered at the order in which some of the lectures are delivered. For example, the student might better understand paranoid states if the lecture on delusions were given before instead of after this lecture. The mechanism of illusions and hallucinations is discussed apart from the study of delusions.

The mechanism of projection and its application to the production of a delusion is explained. The author is of the opinion that, although there may be many causes for a delusional state, its appearance is of grave significance only when it occurs as a manifestation of an abnormal attitude towards life. This abnormal attitude is not merely confined to one subject, but to all departments of life.

An acknowledgement is made to the psychoanalytical school of thought for the elucidation of many psychotic symptoms, although the possibility of harmful results following an attempt to apply Freudian analysis to a psychotic patient is emphasized.

Following the lectures on the psychoses is a lecture on elementary psychopathology. At first sight it might be thought that such a lecture should precede any discussion on mental disorders. It is, however, the author's method of placing the psychoses in a completely different category from that of the neuroses and psychoneuroses. What we call "border line" patients are not really on the border line between sanity and insanity, but are either suffering from true incipient insanity or are psychoneurotics and are never likely to become insane.

The diagnosis in the early stages of the appearance of symptoms may be exceedingly difficult. The question of insight is the deciding factor as to the diagnosis. It is possible that there is a danger in making such a sharp demarcation between the psychoses and the psychoneuroses, as psychoneurotic symptoms may make an independent appearance upon a psychotic basis. It must be remembered also that patients in the early stage of a psychosis often have insight into their condition.

A contrast is made between the methods of treatment of the psychosis and the psychoneurosis. When dealing with the former, the author omits such conditions as general paralysis, arteriopathic and epileptic dementia and confusional states, in which a physical background is present. He confines himself to the so-called biogenic psychosis, manic-depressive psychosis, *dementia præcox* and paranoia. These conditions he considers to be inherent and "incurable" from a psychological standpoint. He points out, however, that many physical ailments are also "incurable", but that clinical knowledge and attention to nursing detail may bring the patient with, say, a chronic nephritis, to a state of useful life. The same principles apply to the management of a psychotic who may be piloted to lead a useful life, though still a psychotic.

The psychoneuroses and neuroses, on the other hand, are definitely curable if the patient is given appropriate treatment. The neuroses (by which he includes the psychoneuroses) are divided by the author into anxiety states, neurasthenia, obsessional neurosis and hysteria.

As regards treatment of the neuroses, Freudian analysis, according to the author, is the only method by which certain neuroses can be cured. Other forms of psychotherapy play their part, and he discusses treatment by suggestion, persuasion and hypnosis. It is agreed that only certain cases are suitable for psychoanalytical methods, and that incalculable harm may be done by such methods in wrong hands. Psychoanalysis should be attempted only by the trained specialist in this particular form of therapy, and all forms of psychotherapy should be a post-graduate study.

The author lays down simple and useful rules by which patients with neuroses can be helped in the course of general practice.

The lectures are attractively written in a terminology that can easily be understood by the psychiatric novice. Controversial points are perhaps glossed over, and the author is somewhat dogmatic in some of his statements, giving a slightly more clear-cut view of mental illness than is really possible. These lectures present a useful jumping-off ground from which the practitioner or student can tackle the more intricate problems of psychiatry from a wider angle.

A MEDICAL HAND BOOK.

IN the opening words of the preface to his "Handbook of Medicine for Final Year Students", Dr. G. F. Walker ventures to claim for it "a high degree of utility and originality". As far as originality is concerned he has certainly achieved success in some of the sections. The book is intended to add to the class of those presenting medicine in a nutshell. It includes advice on "clinical approach" or clinical methods of physical diagnosis. This advice is followed by miscellaneous sections devoted to short descriptions of abdominal pain, diseases of the liver and bile ducts, coma, diseases of the arteries, "blood diseases, certain", and so forth. The best of these sections is that on abdominal tumours, but they are mostly inadequate both in regard to subject matter and description.

The well known book on clinical methods by Hutchison and Rainy (now Hunter) has set a standard in English hand books which most authors will find it impossible to attain. The average student will find it unwise or even dangerous to follow summaries of medicine unless they are prepared by himself from his larger text books. Any writer who in such a short compass and in lengthy sentences sets out to cover present day medicine, even in regard to its pathology and differential diagnosis, is foredoomed to failure.

The most valuable feature of this book is its scattered maxims: "Do not treat old symptoms with new remedies." "Absence of signs of organic disease does not constitute evidence of hysteria." "In framing a diagnosis probability must be accorded a high place." Expressions such as "venulousness" and "normalcy" are at least readily comprehended, while the reader is permitted a little relaxation on reading: "While feeling the pulse absorb all that the hand has to offer", and "the kidneys may be mutually mobile or they may be proptosed". There are some statements which are contrary to clinical experience, such as "albuminuria may be found only in the excitement of a medical examintion" and "a low renal threshold is extremely rare". In a book such as this an author should rigidly exclude anything controversial or even doubtful. In short, we cannot recommend this book to busy final year students.

[1] "Handbook of Medicine for Final Year Students", by G. F. Walker, M.D., M.R.C.P.; 1931. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 255. Price: 10s. 6d. net.

The Medical Journal of Australia

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PROPRIETARY MEDICINES IN VICTORIA.

MAKERS of proprietary medicines may be divided into two groups. One group includes those who are at all times ready to declare the composition of their preparations, and the other includes those who for various reasons prefer to sell preparations which must be regarded as "secret remedies". Medical practitioners have found that certain proprietary preparations are invaluable, and prescribe them for patients, either because the preparations are more elegant or because the drugs contained in them have a more desirable action than those which have received pharmacopœial recognition. Of course, there are practitioners who have got into the habit of prescribing proprietary drugs through sheer laziness. To many proprietary remedies no exception can be taken. That certain of them are accepted for advertisement in this journal is evidence of our belief in their purity and in the justness of the claims made for them. When the proprietors of medicines are not compelled to disclose the drugs for which therapeutic action is claimed, there is danger of worthless or even harmful preparations being sold to the public. The public must be protected from this danger; it must also be protected from its own readiness to be deceived. Other dangers to be remembered are those of self-diagnosis and self-treatment; if non-medical persons diagnose their own illnesses and undertake their own treatment, many pathological conditions will be allowed to advance to a stage at which correct treatment will be of no avail.

In Victoria the *Food and Drugs Standards Regulations* 1930 contain the following clause (Subsection 5 of Section 75):

> Every package containing a patent or proprietary medicine shall have attached thereto a label in which shall be inserted legibly and prominently in English or Latin the names of the drugs therein which have any therapeutic action.

With the passage of the *Pure Food Act* 1905 a Foods Standards Committee was appointed. The Chief Health Officer of the State is Chairman of this Committee, and there are two other medical members, Professor W. A. Osborne, of the Department of Physiology of the University of Melbourne, and Dr. John Dale, Medical Officer of Health of the City of Melbourne. This Committee, in the *Health Act* 1928, is given considerable power in regard to the making of regulations. The Act states that the Governor in Council on the recommendation of the Foods Standards Committee may make regulations; and then follow many clauses describing the type of regulation which may be made. Included in these is the prescribing of standards for the composition or strength or purity or quality of any food or drug, the prescribing of forms or kinds of label to foods or drugs, the exemption of packages from provisions regarding labelling, and so forth. The Governor in Council need not make any regulation recommended by the Committee, and no regulation can be submitted to the Governor in Council until it is approved by the Minister. It is not necessary to describe in detail the other provisions of the Act regarding these regulations, but it is important to note that Parliament cannot veto them. Until December, 1931, regulations made by the Governor in Council on the recommendation of the Foods Standards Committee could be revoked only on the recommendation of the Committee. In December, 1931, the *Health Act* 1931 was passed. Clause 15 of this Act reads as follows:

At the end of section two hundred and fifty-seven of the Principal Act there shall be inserted the following sub-section:

(4) Without prejudice to any other method of revoking any regulation under sub-section (1) of this section, any such regulation whether made before or after the commencement of the *Health Act* 1931 may be revoked by order of the Governor in Council.

It will at once be evident that the Foods Standards Committee has, by the *Health Act* 1931, been shorn of a great deal of its power to protect the public. Subsection 5 of Section 75, quoted above, is one of the regulations adopted by the Governor in Council on the recommendation of the Foods Standards Committee. It may be revoked practically at the will of the Minister. The clause, as it stands, is an admirable safeguard to the people of Victoria; its revocation would be a calamity. Fortunately, the present Premier of Victoria is a medical practitioner, held in high regard by the members of his profession. In view of his medical training, he would be quite unlikely to permit any action of the Governor in Council which would render ineffective Subsection 5 of Section 75 of the *Food and Drug Standards Regulations* 1930. We would urge him, in the best interests of the public, to introduce legislation to amend the *Health Act* 1931, especially in regard to Clause 15. If Section 15 is not deleted, Subsection 5 of Section 75 of the *Food and Drug Standards Regulations* 1930 should at least be exempted from its provisions.

Current Comment.

"PLASMOQUINE" AND "ATEBRIN".

THOUGH quinine is a specific against the various parasites of malaria, it has certain notable deficiencies. It has unpleasant, sometimes even distressing effects, and it has little or no influence on either the sporozoites or the gametocytes. That "Plasmoquine" has a pronounced destructive effect on the gametocytes is unquestioned; but its effect on the parasite in its asexual phase is not nearly so powerful as that of quinine. Its value as a prophylactic was discussed in these pages in the issue of March 12, 1932. The introduction of "Plasmoquine" is a step forward in the control of malaria; the next step is the discovery of a drug which is lethal to the asexual parasites and which can take the place of quinine. Possibly "Atebrin", a synthetic drug allied to "Plasmoquine", fulfils the requirements. L. Everard Napier and B. M. Das Gupta have subjected "Atebrin" to a clinical investigation.[1] They conclude that the administration of "Atebrin" in a dose of 0·1 gramme three times a day for four days controls the fever of each of the three forms of malarial infection, effects a disappearance of the asexual forms of the parasite from the peripheral blood, and is neither toxic nor unpleasant to take. "Atebrin" apparently has no effect on the gametocytes and does not prevent their development. Napier and Das Gupta are unable to say whether "Atebrin" is more or less efficacious than quinine. They admit that, even in the doses employed by them, it might be toxic to certain sensitive persons. Napier, Dorothy Butcher and C. R. Das Gupta have investigated the value of the administration of "Plasmoquine" as a prophylactic, and "Atebrin" as a therapeutic agent in the field.[2] They gave 0·01 gramme of "Plasmoquine" three times a week to native Indian boys over twelve years old, and half this dose to younger boys. These doses are considerably smaller than those usually recommended. The most interesting feature of the results of attempted prophylaxis was that the incidence of malaria was considerably higher among the natives who had received "Plasmoquine" than among those who had not. The investigators suggest that "Plasmoquine" in such small doses may have a provocative action. The results of treatment with "Atebrin" and "Plasmoquine" were satisfactory.

C. Strickland and D. N. Roy fed anophelene mosquitoes with blood containing gametocytes of *Plasmodium falciparum* after the patient had received a course of treatment with "Atebrin".[3] Development did not occur in the body of any mosquito fed the day after the cessation of treatment, but occurred in all mosquitoes fed three days after the cessation of treatment. The presumption is that, as the result of "Atebrin" therapy, the gametocytes of *Plasmodium falciparum* are innocuous to the mosquito. It would seem that a person receiving treatment by "Atebrin" is unable to transmit malaria, however many gametocytes he may harbour in his peripheral blood. It should be noted, however, that Strickland and Roy made observations on one patient only.

The information contained in these three papers is interesting and valuable, and should be in the possession of every medical practitioner engaged in the treatment of malaria. The evidence provided is too slight to warrant the conclusion that small doses of "Plasmoquine" may have a provocative effect; still, the suggestion is not so grotesque as perhaps at first it appears; similar suggestions have been made concerning the action of other drugs in other diseases. The most important conclusions are those concerning the value of "Atebrin". This is a drug that may eventually hold an important place in the treatment of malaria; but careful medical practitioners will not yet allow it to supplant quinine. Much more experimental work must

[1] *The Indian Medical Gazette*, April, 1932.
[2] *Ibidem.*
[3] *Ibidem.*

be done. Quinine is a drug of proven value; its deficiencies are known. All that is certainly known of "Atebrin" is that it is lethal to the three species of human plasmodia; its toxicity requires further study.

GUANIDINE AND ALIMENTARY INTOXICATION.

IT has long been known that guanidine may produce serious toxic effects upon the human organism. Noel Paton ascribed tetany to a toxæmia due to excess of guanidines or methyl-guanidine in the blood. These substances produced in laboratory animals manifestations identical with those of idiopathic and post-operative tetany. Guanidine is said to stimulate outgoing spinal neurones and, in small doses, to excite peripheral neuromyones, large doses paralysing them. Dimethyl-guanidine is eight times as toxic as the monomethyl and its toxic effects are identical with spasmophilia. It has been said that the parathyreoids are able directly to detoxicate guanidine, but Noel Paton regarded the parathyreoids as producing an internal secretion controlling muscle tone by regulating its metabolism, that is, the production or destruction of guanidine in the body. Paton said that a fall in calcium salts, especially if the nerve structures concerned in maintenance of tone were sensitized, either by methyl-guanidine or other factor, might play a great part in determining the onset of symptoms and the administration of large amounts of calcium might overcome the effect of methyl-guanidine. But in tetany calcium deficiency was not always present, and in uræmia a fall of calcium may occur without tetany. A fall in blood calcium usually coincides with a rise of blood cholesterol and *vice versa*.

K. Dodd, A. S. Minot and H. Casparis[1] give an account of non-specific diarrhœas in infants, of severe and often fatal type, referred to as alimentary intoxication, but often termed summer diarrhœa, colitis or *cholera infantum*. Parenteral infection (*otitis media* and perhaps mastoiditis), summer heat, dehydration and artificial feeding are important ætiological factors. The babies usually vomit everything. The stools are innumerable and watery. There may be altered blood in the vomit and stools. Air hunger and terminal convulsions may occur. The outstanding chemical changes in the body are those secondary to dehydration and the loss of fixed base in the stools. The total proteins and chlorides in the blood are usually increased and the base bicarbonates diminished. Less constant changes in the non-protein nitrogen, inorganic phosphates and lactic acid have been noted. In certain cases there seems to be an unidentified toxic agent, the action of which persists and prevents recovery even after the subsidence of infection and when attempts to maintain acid base and fluid equilibrium have been continued.

The hot summer of 1930 in Nashville produced almost an epidemic of alimentary intoxication. Results of treatment were discouraging. Many relapsed into acidosis and a large percentage of patients died. An obscure toxic factor was not adequately combated. This factor could injure almost any tissue. The bloody vomit, diarrhœa, acidosis with prostration or nervous hyperexcitability resembled the condition observed in animals with increased guanidine in the blood. In intoxicated animals a frequent accompaniment of hyperguanidinæmia is severe hypoglycæmia. In this epidemic the blood sugar levels were studied in some infants and were sometimes found to be low. In some cases also guanidine determinations in the blood were found comparable to those in intoxicated animals. With the guanidine increase there was no evident rise of creatine or creatinine. The average normal concentration of guanidine in the blood of children and dogs is about 0·35 milligramme per 100 cubic centimetres. In severely intoxicated dogs it varies from 0·6 to 1·5 milligrammes, corresponding to the levels reached in intoxicated children.

An antagonistic action between calcium salts and guanidine has been shown. Animals receiving calcium before or early in the course of intoxication can be prevented from developing symptoms of poisoning in spite of high blood guanidine concentration, and can be cured by persistent calcium therapy. Five of the children were thus treated and three recovered. The number was too small to warrant any conclusions, but there is evidence that guanidine or a similar substance in summer diarrhœa may be very toxic. The source or fate of guanidine in the body is not known. Because of the presence of the guanidine group in the amino-acid arginine and in creatine, it is thought that these substances may be related. Guanidine, except in slight traces, is a pathological constituent of the blood. The colorimetric method used does not distinguish between different forms of guanidine. We may be dealing with guanidine or the more toxic methyl-guanidine. The infants concerned gave symptoms and tissue changes very similar to those of experimental guanidine poisoning. Previous investigations associated guanidine increase with liver injury or parathyreoid deficiency. In the cases here reported there is no evidence that such factors were primary. Other studies seem to show that necrotic or autolysing tissues may be a source of guanidine. This may be related to severe dehydration. Guanidinæmia is not influenced by calcium, even when relief of symptoms has been obtained. But if a high guanidine level continues for long without the inhibiting action of extra calcium, irreparable tissue changes may result and cause death. In these circumstances the intramuscular injection of calcium gluconate is recommended.

This work is of interest when considered in conjunction with that of Major on the effect of guanidine bases on blood pressure, and of Watanbe on the effect of guanidine on the blood sugar. Guanidine is too toxic for therapeutic administration and the use of its derivatives has been abandoned.

[1] *American Journal of Diseases of Children*, January, 1932.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Functional Uterine Bleeding.

D. J. CANNON (*The Journal of Obstetrics and Gynæcology of the British Empire*, Autumn Number, 1931) has set out to analyse the nature and present a logical classification of functional uterine bleeding in the light of the most recent physiology of menstruation. The author means by functional bleeding those severe bleedings due to disturbances in the factors intrinsic to the menstrual process itself, excluding those which are due to uterine pathological conditions or gross anatomical lesions. He discusses the subject under two headings: functional uterine bleeding due to ovarian dysfunction, and *menorrhagia simplex*. He subdivides the former into two groups, the first of which is *metropathia hæmorrhagica* (Schroeder). This may occur at any period of the reproductive cycle, but mainly about the time of the menopause. The patient has continuous bleeding perhaps for one or two months or longer. It may be preceded by amenorrhœa of six to twelve weeks' duration. This type is sometimes called metrostaxis. Microscopical examination in these conditions reveals hyperplasia of both the epithelial and stromal elements. There is invariably fibrocystic disease of either one or both ovaries. There may be one or two or even three cysts. Wilfred Shaw claims that in these cases there is evidence of diminished follicular activity. The most characteristic feature is the absence of a *corpus luteum*. When a *corpus luteum* it present it is always immature. According to Wilfred Shaw, the hyperplasia is a primary condition, the cause of which is obscure; the ovarian changes are secondary to a hormone manufactured in this endometrium. The second type of this group is associated with epimenorrhagia, which means not only increased loss of blood, but also a shortening of the menstrual rhythm. This symptom is associated with premature rupture of the follicle and premature degeneration of the *corpus luteum*. In these cases there appears to be an increase in the number of cystic follicles, which, however, do not grow to the size of follicular cysts. Wilfred Shaw calls this second type "hyperovarian", to distinguish it from the first type, which he calls "hypoovarian". The author, however, prefers to use the terms "hypofollicular" and "hyperfollicular". The second group of bleedings consists of those cases which come under the heading of *menorrhagia simplex*. About 15% do not fit into either classification. Menorrhagia is the outstanding symptom. In some cases the bleeding is very severe. The uterus may appear normal on examination, or a little hard and firm. There is no evidence of hyperplasia. Not long ago all cases of severe bleeding not due to conditions following labour or abortion or gross pathological lesions were attributed either to chronic endometritis or chronic metritis. The author points out that in the light of recent research it is known that endometritis is relatively rare. Many conditions previously classified as chronic endometritis were really due to hyperplasia. Chronic endometritis may cause discharge, but there is no proof that it causes excessive bleeding. The author reviews the work of Whitehouse, who seems to regard atrophy of the endometrial stroma as the anatomical basis of this type of uterine bleeding. He gives an interesting summary of Whitehouse's views, with which he does not entirely agree. *Fibrosis uteri* or chronic metritis has been regarded as one of the causes, and this is the British point of view. The German school entirely disagrees with this, and Fletcher Shaw in his recent work agrees with the German investigators. Apparently the presence of connective tissue in the musculature of the uterus is not regarded as sufficient cause for bleeding from the endometrium, and other causes should be looked for. In many cases, however, constitutional causes are present which may account for the bleeding. Disordered calcium metabolism is one. Disturbance of the thyreoid gland is another common cause of excessive bleeding which should always be borne in mind, and, of course, in every case it is essential to exclude conditions such as submucous fibro-myomata, carcinoma, and conditions following labour or abortion.

Malignant Ovarian Neoplasms.

CHARLES C. NORRIS AND DOUGLAS P. MURPHY (*American Journal of Obstetrics and Gynecology*, June, 1932) reports the end-results in a series of 93 cases of malignant ovarian tumour. In every case the diagnosis had been established microscopically. Of 125 patients, 13 died within 30 days of operation. Of 93 patients treated for three years or more, 50 were apparently cured. The glandular type of ovarian carcinoma was nearly twice as frequent as the papillary type; 34% were bilateral. All types of tumour had approximately the same tendency to growing on both sides. The authors conclude that even patients with advanced growths should be operated on, as in some cases it is found that the condition is not malignant and can be relieved by operation. They consider that both ovaries and the uterus should be removed in every case, even though the other ovary appears normal at the time of operation.

Menorrhagia due to Hypothyreoidism.

W. C. WATERS AND GEORGE A. WILLIAMS (*American Journal of Obstetrics and Gynecology*, April, 1932) discuss the influence of hypothyreoidism on menorrhagia. After a brief review of the literature they report a series of five cases in which there was profuse menstruation without any definite pelvic disturbance. All five patients responded to treatment with thyreoid gland. The authors are of the opinion that in conditions of hypothyreoidism it is far more common for excessive menstrual bleeding rather than diminished bleeding to occur; amenorrhœa is very rarely associated with diminished function of the thyreoid gland. Whenever a patient has excessive menstruation and no definite disturbance of the pelvic organs can be found on clinical examination, they urge that thyreoid gland therapy should be tried before operative measures are instituted. It is not necessary to find an alteration in the basal metabolic rate in order to establish the presence of hypothyreoidism. If the patient responds to small doses of thyreoid, as they frequently do, the condition is established as a clinical entity. It would appear to be much commoner than is generally reported.

Endometrial Hyperplasia and Carcinoma of the Body of the Uterus.

HOWARD C. TAYLOR (*American Journal of Obstetrics and Gynecology*, March, 1932), at the Roosevelt Hospital, New York City, carried out a series of investigations on the apparent transformation of hyperplasia to carcinoma. He investigated material from four hundred and fifteen cases. These included cases of hyperplasia of the endometrium, endometrial polypi, carcinoma of the corpus, and conditions incorrectly diagnosed as hyperplasia. The author has studied the relationship of the two conditions from both an ætiological and a clinical aspect. In at least six instances definite evidence has been reported of a change in the character of the tissues obtained by successive curettings from the same patient from hyperplasia into carcinoma. In one hundred and twenty-two cases of carcinoma it was shown that most of the women had at some time before their final operation for cancer been under treatment for abnormal uterine bleeding. In five cases there was an association of diffuse hyperplasia and carcinoma in the same uterus. From a clinical point of view, the author concludes that all patients with postmenopausal bleeding should be investigated with a curette, and in every instance the scrapings should be examined microscopically. It is quite easy to miss a small carcinoma in its early stage with the curette, and he concludes finally that all cases in which there is marked hyperplasia should be regarded as precancerous and treated as such, either with radium or by hysterectomy.

Anterior Pituitary Sex Hormones.

S. D. SOULE (*American Journal of Obstetrics and Gynecology*, May, 1932) has studied anterior pituitary hormones as detected by the original technique of Aschheim and Zondek, and reports his conclusions as the

result of two hundred experiments. Various animals and different fluids, such as urine, blood serum, blood plasma *et cetera*, have been used. The present technique is to use rabbits weighing between 1,500 and 1,600 grammes, and detoxicated blood serum. With this technique a reaction was well developed before forty-eight hours. In regard to abortion, the test failed to yield a reaction five to seven days after complete emptying of the uterus. As an aid to clinical diagnosis, a positive result of a test after this time is the indication of the diagnosis of retained tissue. The author believes that the test is reliable and that rabbits of 1,500 grammes are the most suitable animals and blood plasma the best material.

OBSTETRICS.

Bacillus Welchii Infection of the Uterus During Labour.

E. M. CROOKS (*The Chinese Medical Journal*, February, 1932) reports seven cases of *Bacillus welchii* infection of the uterus. Most patients were admitted to hospital after days of labour and repeated examinations by Chinese midwives, who do not even bother to wash their dirty hands before making an examination. In two instances the position was transverse and the arm had been pulled by the attendants. No antiserum was available. The treatment varied from simple extraction to Cæsarean section with hysterectomy and suture of the uterine stump to the abdominal wall. The author has found the bacillus in the stools of some of these patients. This would provide a simple explanation of the introduction of the organisms into the uterus by manipulative interference. Whenever a patient is admitted with a history of long labour and any form of interference, and the fœtus is dead, the possibility of this infection must be thought of.

Non-Fixation of the Head in Primiparæ.

O. PANKOW (*Monatsschrift für Geburtshülfe und Gynäkologie*, January, 1932) found that in 13·3% of a series of 1,959 *primparæ* the head was not fixed at the onset of labour. In two-thirds of the cases this was associated with contracted pelvis. In other words, 4·5% of all the *primiparæ* showed non-fixation of the head, despite normal measurements, both maternal and fœtal. In nearly 60% the membranes ruptured prematurely, but the course of the subsequent labour varied greatly. The mechanism of labour was not altered, despite the frequent occurrence of uterine inertia. The incidence of forceps delivery was three times as great as with all deliveries in the hospital. He emphasizes that all such patients should be treated in hospital because of the operative incidence. In private practice the use of uterine stimulants and the too early resource to forceps tend to cause severe damage to both mother and child.

Induction of Abortion.

F. ENGELMANN (*Deutsche Medizinische Wochenschrift*, January 29, 1932) discusses the newer method of induction of abortion by the intra-uterine injection of pastes. He states that this procedure has been largely used by the general public. The pastes usually consists of a mixture of iodine, potassium iodide, essential oils and small doses of pituitary extract. The original pastes contained fatty substances, but recent formulæ are stated to be fat-free and to contain no air bubbles. The literature of the subject is summarized and the effect on the uterine mucosa as well as on the ovum is described. So far seventeen deaths have been reported; in the majority the fatal issue was due to fat or air embolism. The author is strongly of the opinion that the use of such remedies is far from safe and that medical practitioners should not use them.

"Nembutal" and Chloral Hydrate in Child-Birth.

J. V. O'SULLIVAN AND W. W. CRANER (*The Lancet*, January 16, 1932) record sixty consecutive cases of labour treated by them in the London Hospital, in which "Nembutal" (sodium-ethyl-methyl-butyl barbiturate) and chloral hydrate were administered as analgesics. "Nembutal" is put up in gelatin-coated capsules (one and a half grains in each), as it undergoes hydrolysis on being kept. Chloral hydrate is administered in 2·0 gramme (thirty grain) doses in 900 cubic centimetres (three ounces) of fresh, home-made lemonade, sweetened with three or four teaspoonfuls of sugar. A total dose of 0·45 gramme (seven and a half grains) of "Nembutal" and 7·2 grammes (one hundred and twenty grains) of chloral hydrate should not be exceeded in twelve hours. The drugs are not given simultaneously; the "Nembutal" should always precede the chloral by ten minutes to avoid vomiting. The initial doses are 0·18 gramme (three grains) of "Nembutal" and 2·0 grammes (thirty grains) of chloral hydrate. The subsequent doses are 0·09 gramme (one and a half grains) of "Nembutal" and 2·0 grammes of chloral hydrate. The first "repeat" dose is given after two hours and the remaining doses every three. The interval between doses should never exceed four hours. The first dose is given in *primiparæ* when the *os uteri* is two-fifths to three-fifths dilated, in *multiparæ* when the os is one-fifth to two-fifths dilated and regular pains are present. The authors have found no contraindications to the use of these drugs. They refer in detail to cases in which this combination of drugs has been used. They consider that the drugs might be administered by midwives, provided that they follow the technique carefully and never vary it without the direct permission of a medical practitioner.

Treatment of Pyelitis of Pregnancy.

H. NAUJOKS (*Deutsche Medizinische Wochenschrift*, February 26, 1932) reviews the various theories regarding the ætiology of pyelitis of pregnancy and considers that it is due to an atony of the ureters caused by some toxic state of pregnancy. He summarizes various experiments with "Tonephin", a preparation containing the pressor principle of the posterior pituitary lobe. His investigations show that an injection of five units produces contractions of the ureters within ten to fifteen minutes. No ill effects have been noted, especially in regard to inducing uterine contractions, even when fifteen units have been given within a short period. Naturally, the older methods of treatment with alkaline drugs and diet are still combined with five to ten units given intramuscularly each day. The author considers that this pressor principle of pituitary extract is a valuable aid in the treatment of pyelitis.

Mechanism of Delivery.

W. LÜTTGE (*Monatsschrift für Geburtshülfe und Gynäkologie*, January, 1932) maintains that the usual text book description of the initial descent of the head into the pelvis is wrong. According to him, the lowest part of the fœtal head which engages at first in the pelvic brim is the parietal bone and not the region of the posterior fontanelle and occiput. The first pressure applied to the presenting part is a lateral one, forcing the parietals downwards. Later pressure on the fœtus acts through the spinal column, driving the occiput to the lowest level. The latter is generally considered to be the initial movement in descent and flexion of the head, but from the radiograms which he presents, the author is convinced that lateral pressure precedes one of *vis a tergo* and produces in normal cases the initial phenomena of the early stages in the mechanism of delivery.

Abdominal Palpation During Labour.

W. HAUPT (*Monatsschrift für Geburtshülfe und Gynäkologie*, February, 1932) refers to the importance of restricting vaginal examinations during labour as far as possible. Valuable information can be obtained by observing the descent of the fœtal head by means of abdominal palpation. He describes the modification of the fourth grip of Leopold, first elaborated by Gauss. Gauss carefully locates the position of the chin. When the head is fully fixed the chin can be felt nearer the mid-line than is usually expected. Difficulty in detecting the chin may be caused by a rigid abdominal wall, obliquity of the parietal bones, or in some cases by an arm covering the face. The article is illustrated with radiograms taken during labour.

British Medical Association News.

MEETING OF THE FEDERAL COMMITTEE.

A MEETING OF THE FEDERAL COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA was held at the British Medical Association House, 135, Macquarie Street, Sydney, on September 1, 1932, DR. J. ADAM DICK, the Acting Chairman, in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales: Dr. J. Adam Dick, C.M.G., Dr. C. H. E. Lawes.

Queensland: Dr. D. G. Croll, C.B.E.

Tasmania: Dr. Gregory Sprott, Dr. E. Brettingham Moore (as substitute for Dr. A. W. Shugg).

Victoria: Dr. F. L. Davies, Dr. R. H. Fetherston (as substitute for Dr. J. Newman Morris).

Dr. C. H. E. Lawes acted as proxy for Dr. F. A. Hadley, of the Western Australian Branch. Dr. J. Adam Dick acted as proxy for Dr. Bronte Smeaton, of the South Australian Branch. Dr. D. G. Croll acted as proxy for Dr. E. S. Meyers, of the Queensland Branch.

Minutes.

The minutes of the previous meeting of February 16 and 17, 1932, copies of which had been sent to members after the meeting, were submitted and signed as correct.

Financial Statement.

The financial statements for the six months ended June 30, 1932, prepared and audited by Messrs. Coates, Cunningham and Company, Chartered Accountants, together with the Australasian Medical Congress (British Medical Association) accumulated funds account, were presented and adopted.

Finance.

The amount of the contribution which should be paid by the Branches to the general expenses of the Federal Committee was discussed. Dr. C. H. E. Lawes, the Honorary Secretary, pointed out that the Federal Committee had power to levy a *per capita* payment of two shillings *per annum*. It was decided that a *per capita* payment of one shilling should be made for the ensuing twelve months. On the motion of Dr. D. G. Croll, seconded by Dr. Gregory Sprott, it was resolved:

> That at the request of any Branch the Federal Committee may authorize suspension of payment until such time as the money is required.

Medical Officers' Relief Fund.

The balance sheet and revenue account of the Medical Officers' Relief Fund (Federal), together with recommendations of the Trustees, were submitted and adopted. Dr. George Armstrong and Dr. W. H. Crago forwarded their resignations as Trustees. It was resolved that both resignations be accepted with regret, and that Dr. Armstrong and Dr. Crago should be thanked for their many years of service. Dr. G. C. Willcocks, O.B.E., and Dr. J. Adam Dick, C.M.G., were appointed to fill the vacancies.

Accident Insurance in Western Australia.

The Honorary Secretary read a letter from the Western Australian Branch asking for advice in regard to a proposed agreement with an insurance company for accident, sickness and fire policy cover for members of the Branch. It was noted that the insurance company undertook to accept proposals for insurance from members of the Branch at a low rate of premium if the Branch Council would notify members of the arrangement and advise them to effect insurance with the company in question and with no other.

After discussion it was resolved:

> That the Federal Committee would suggest that the Western Australian Branch Council obtain a legal opinion as to whether the proposed agreement would infringe the local insurance act as it would in Queensland; and that the Branch be advised also that the Federal Committee does not approve of a Branch Council entering into such business relations, which are better left to an affiliated insurance body, such as the British Medical Agencies of New South Wales and Victoria.

Centenary Meeting of the Association.

The Honorary Secretary reported that he had on July 15, 1932, sent a radiogram to the Parent Association conveying the congratulations of the Federal Committee on the occasion of the centenary of the Association.

Subscription from Overseas Branches.

At the previous meeting of the Federal Committee the question of a reduction of the annual subscription paid by the Branches in Australia to the Parent Body had been discussed. It was decided at that meeting that the Chairman of the Committee, Sir Henry Newland, and the Vice-Chairman, Dr. J. Newman Morris, should discuss the matter with the Medical Secretary of the Association while they were in England. The Honorary Secretary reported that he had received a letter from Sir Henry Newland to the effect that the matter had been discussed with the Council of the Parent Body, that Dr. T. P. Dunhill and Professor R. J. A. Berry had joined him (Sir Henry Newland) and Dr. Newman Morris in making representations to authorities of the Parent Body, but that no finality had been reached. It was resolved that further discussion be postponed until the next meeting.

Dr. Alfred Cox Testimonial Fund.

A letter was read from Dr. E. R. Fothergill, the Honorary Secretary of the General Committee of the Dr. Alfred Cox Testimonial Fund, acknowledging the receipt of a draft of £20 6s. 9d., representing £25 10s., less exchange *et cetera*, collected from members in Australia for the testimonial.

British Medical Association Scholarships and Grants.

It was noted that an application had been received from a medical practitioner in Australia for a British Medical Association scholarship and that the papers had been forwarded to the Deputy Medical Secretary of the Association.

Federal Council.

At the previous meeting of the Federal Committee a letter from the Under-Secretary of Justice, of the Department of the Attorney-General and of Justice of New South Wales, was considered. This letter had been sent to Messrs. Tress and Cocks, Solicitors of the Federal Committee, and dealt with the proposal registration of the Federal Council under the provisions of Section 52 of the *Companies Act*, 1899, of New South Wales. The Under-Secretary set out in a schedule certain amendments to the Memorandum and Articles of Association, and indicated that the issue of a licence would be considered when these amendments were embodied. At the previous meeting Dr. J. Adam Dick and Dr. C. H. E. Lawes were appointed a subcommittee to interview the Under-Secretary.

Dr. J. Adam Dick reported that he and Dr. Lawes acted with Mr. Herbert Tress, of Messrs. Tress and Cocks, in the matter, and Dr. Lawes placed before the Committee the Memorandum and Articles of Association amended according to the requirements of the Under-Secretary. At the invitation of the Federal Committee, Mr. Herbert Tress attended the meeting and explained the legal position. The Committee also had before it a letter from Mr. Tress, dated July 16, 1932, in which the position was discussed. Mr. Tress expressed the view that three courses were open to the Federal Council: (i) To accept the modified constitution and proceed to incorporation under it. (ii)

To incorporate a company under the provisions of the *Companies Act* of New South Wales, using the word "Limited" as part of the name, in which event most of the objections raised by the Crown Solicitor might be disregarded. (iii) To continue as an unincorporated body under the present constitution of the Federal Committee or such amended constitution or rules as might be adopted from time to time with the sanction of the Branches and of the British Medical Association.

Mr. Tress discussed with members of the Committee the advantages of incorporation. He pointed out that incorporation would be necessary only if the Federal Council intended to own property or to engage in other specified activities. He also pointed out that if the Council were incorporated, the liability of its members would be restricted in certain directions.

It was resolved, on the motion of Dr. D. G. Croll, seconded by Dr. F. L. Davies:

> That the Federal Committee be incorporated and that the amendments to the proposed Memorandum and Articles of Association required by the Minister for Justice be approved.

It was further resolved, on the motion of Dr. F. L. Davies, seconded by Dr. R. H. Fetherston:

> That the name of Charles Herbert Essery Lawes be substituted for that of Robert Henry Todd in Section 41 of the Articles of Association as the first Secretary of the Federal Council.

Annual Meeting of the Association.

The following motion was submitted as a matter of urgency by the Victorian Branch:

> That representations be made to Sir Henry Newland to approach the Parent Association to see if it would be possible to hold the annual meeting of the British Medical Association in Victoria in 1935, the occasion being the centenary of the City of Melbourne.

The Federal Committee decided to accede to the request.

Australasian Medical Congress (British Medical Association).

A letter was read from Dr. D. D. Paton, President-Elect of the fourth session of the Australasian Medical Congress (British Medical Association), in which reference was made to the date of holding the fourth session. Dr. Paton reported that the Executive Committee of Congress had met on April 11, 1932, for the purpose of discussing the recommendation of the Federal Committee at its previous meeting, that the fourth session should be postponed for one year. Dr. Paton reported that the Executive thought it advisable to recommend to the Federal Committee that the fourth session, if held in Perth, should not be held before October, 1934. He added that if the Federal Committee decided that it was not in the best interests of the British Medical Association in the Commonwealth and New Zealand to be so long without a congress, the Committee should feel itself at liberty to recall its decision and arrange for a congress to be held in one of the more central States. If the Congress was withdrawn from Perth, the Executive Committee in Perth hoped that the Western Australian Branch would be given the opportunity of coming in again at the first succeeding vacancy.

In the discussion on Dr. Paton's letter the view was expressed that it would be a mistake to allow the Congress to lapse for too long a period, and that medical practitioners would be likely to lose the "congress habit". It was also evident that the Branches in some of the other States were eagerly awaiting their turn to hold a congress.

It was resolved, on the motion of Dr. D. G. Croll, seconded by Dr. Gregory Sprott:

> That the Federal Committee feels that any further postponement beyond 1933 is not in the interests of the profession, and that if the Executive Committee of Congress does not find it practicable to hold a congress by that date, a congress should be held as early as possible in another State.
>
> That no promise can be made as to the dates of future congresses.

Conditions of Service of Medical Officers at Nauru.

At its previous meeting the Federal Committee discussed the conditions of service of medical officers at Nauru. It was reported that a new medical officer had been appointed for service at Nauru and that the conditions of service were unchanged. It was resolved that no further action be taken.

The Naval Medical Service.

Further reference was made to the conditions of service of medical officers in the Royal Australian Navy. This question was discussed by the Federal Committee at its last meeting. At that meeting it was resolved that the Acting Chairman and the Honorary Secretary should approach the Minister and that the Editor of THE MEDICAL JOURNAL OF AUSTRALIA be asked to deal with the matter. This subject was discussed in the editorial pages of this journal on April 9, 1932. The Acting Chairman reported that, in company with the Honorary Secretary, he had interviewed the Honourable Joseph Francis, Assistant Minister for Defence, and had put the views of the Committee before him. The Honorary Secretary read a letter which he had received from Senator G. F. Pearce, Minister for Defence. The letter of the Minister was regarded by the members of the Committee as unsatisfactory, and it was left to the Acting Chairman and the Honorary Secretary to write a letter of protest to the Minister. This matter will be referred to at a later date in this journal.

A memorandum was read from the Honorary Secretary of a conference held on July 28, 1932, to consider the question of medical attendance on injured dockyard workers. It was pointed out that under existing arrangements civilian workers at the dockyards were being attended by naval medical officers. The remuneration of naval medical officers for this service was not adequate, and patients were being taken from their regular medical attendants. It was resolved that a report on the matter should be prepared and presented at the next meeting of the Federal Committee.

Permanent Commonwealth Medical Officers.

A request was received from the Queensland Branch that a protest be made by the Federal Committee against the retirement of permanent medical officers of the Commonwealth Department of Health. It was held that medical officers had been retired without compensation and that in some instances officers had been offered work for which their training made them unsuitable. It was resolved that a letter should be sent to the Minister protesting against the retirement of any permanent medical officer of the Commonwealth Department of Health without compensation, and also that a protest be made against the curtailment of the work of the Division of Tropical Hygiene since it was an essential Commonwealth activity. It was thought that if retrenchment were necessary, the retrenchment should be made in work that could be done by the States.

Repatriation Department.

Reference was made to the arrangement made in 1924 by medium of the Federal Committee and the Branch Councils for medical officers of friendly societies to attend widows and orphans of soldiers whose death was the direct result of war service, and the widowed mothers of such deceased unmarried soldiers. It was pointed out that the Repatriation Department held that this arrangement covered deaths due to war service, whenever the death occurred. The Victorian Branch asked that an inquiry be made in regard to the qualification of beneficiaries under this scheme. Members of the Committee held that the arrangement covered a definite list of those who had died before 1924. They thought that the Department was responsible for the medical attendance on dependants of

soldiers who died after that year, and that the Department should shoulder its own responsibility. The medical profession had agreed to take over the attendance of the initial lot only. It was resolved that Dr. F. L. Davies and Dr. R. H. Fetherston should interview the Repatriation Department and report at the next meeting.

Thanks.

Votes of thanks were accorded to the Acting Chairman, Dr. J. Adam Dick, for presiding and for entertaining members at luncheon, and to the Council of the New South Wales Branch for their hospitality and for the accommodation provided for the meeting.

Date and Place of Next Meeting.

It was determined that it be left to the Acting Chairman to fix the date and place of the next meeting.

Correspondence.

THE FUTURE OF MEDICINE AS A PROFESSION.

SIR: Could you find space in your columns for some remarks on the future of medicine as a profession?

The medical profession in France is very much troubled about the increasing ratio of practitioners to population, at present one to 1,500 approximately. Steps have already been taken, and others proposed, to deal with the overcrowding.

But, if the situation gives cause for alarm in France, what of conditions elsewhere? In the British Isles the figures for 1931 (taken from the "Medical Directory" and "Whitaker's Almanack") show a ratio of one to 1,083. In New South Wales the figures for 1911 (taken from the "Official Year Book" and from the *Government Gazette*) were one to 1,031. The figures for 1930 (taken from the Statistical Register and from the State "Year Book") show the astonishing ratio of one to 780.

The 1930 figures in the other Australian States are as follows: Queensland, one in 1,829; South Australia, one in 1,257; Western Australia, one in 822; Tasmania, one in 777; Victoria, one in 704.

What of the future in New South Wales? It is possible to predict, from the following data, that the relative number of practitioners will become greater and greater. (i) The curve up to the present has shown a steep upward gradient, especially in recent years. (ii) The numbers of students recently admitted to the first year in medicine are as follows: 1926, 58; 1928, 68; 1930, 99; 1931, 129; 1932, 148. (iii) The rate of growth of population is rapidly declining.

This relative increase in the number of practitioners is accompanied by an increasing tendency on the part of the people to seek public hospital attention—a tendency easily verified by referring to the annual reports of any of our metropolitan hospitals, and comparing the outpatients statistics for 1920 and 1930. The increase varies for different hospitals between 50% and 300% during these years. Will these patients return to their private doctor when they can afford to do so? We do not know.

The natural outcome of these tendencies is foreshadowed in the central European countries. Amongst practitioners there, the best situated are those who hold positions in institutions where they work hard and receive their keep and a small pittance. In private practice the most eminent men receive but little remuneration for their services and are pleased to get comparatively small fees for coaching visitors from abroad. I personally knew of one, a famous man, who was asked to lecture in America, but who could not afford the passage money which was eventually paid by his American admirers. So much for the belief that "there is always room at the top". The rank and file are in a deplorable condition and worse off than the labourer who is cared for by a socialistic government. In Hungary the ratio of practitioners to population is one in 1,100, and most of the doctors are in Budapest, where the ratio is one in 324. Seeing that 82% of the people are socially assured and must consult the official doctor, it follows that the *clientèle* of the private practitioners is represented by 58 units per head. (*Bulletin professionnel des Médecins suisses*; quoted by *Journal de Médecine de Bordeaux*.)

No doubt competition is a good thing in moderation, but too much can act only detrimentally to the profession by developing the importance of the economic and commercial side to an unpleasant degree. The public would suffer as well as the profession. "A hungry doctor is a menace."

Moreover, it is apparent that many of the young people at present studying medicine are doomed to disappointment. They are studying medicine because there is no work elsewhere and the study solves the problem of giving them something to do for the next six or seven years. But what then? When they have completed their course many will suffer the degenerating influence of semi-idleness during the best years of their lives—in fact, for the whole of their lives, if the present tendencies continue.

Meantime, business and commerce will improve as the world straightens things out for itself and the non-medical young people will again be able to put their shoulder to the wheel and find adequate use for their energies. But the natural law of supply and demand can work but slowly in medicine, for young people with a medical training are for the most part unfitted for anything else. The factor in happiness with which I am now concerned is not altogether a financial one, but rather the necessity for adequate occupation for vigorous and highly trained brains.

I believe that attention should be drawn to the above facts in order that something may be done. There are remedies. One of the most important is that the youths about to map out their future, and the parents of such youths, should not be allowed to enter the long corridor of medical education except with their eyes thoroughly open and nursing no delusions as to probable conditions at the other end.

The limiting by competitive examination of the numbers entering the faculty is another possible remedy which, together with others, deserves very careful consideration.

Yours, etc.,

VICTOR J. KINSELLA.

171, Macquarie Street,
Sydney,
July 25, 1932.

DIATHERMY OF TONSILS.

SIR: In your leader "Diathermy of Tonsils", August 6, 1932, concluding sentence of last paragraph but one, you say: "The medical practitioner who finds it necessary to consider how little harm he can do with his operative interference (properly so called) had far better leave the field to others."

This sentence has to be read with preceding matter, of course, but even so it seems to me to be contrary to the true spirit of surgery. Unless an operation is one of urgency, actually a measure to save life, the surgeon should always ask himself the two questions together, namely: "Can I do any good by operating?" and "Am I likely to do any harm by operating?" Having satisfied himself on the former the latter must be faced and should be faced frankly.

To my mind more harm comes from neglecting this very important question than possibly any other in surgical practice.

I must therefore very definitely join issue with you, Sir, on this point. I would almost reverse this sentence as a general truth and say: "The medical practitioner who does not consider how little harm . . . had far better leave the field to others."

On the subject proper of your article I know nothing, but would like to make this comment. The argument about diathermy of tonsils has seemed to me to miss the point very often. Because a man has a diathermy machine he is not therefore, as you say, a proper person to remove tonsils for exactly the same reason that the man who has a scalpel is not therefore capable of doing a major surgical operation.

Your remarks *re* pharmacists and Dr. Hamilton's letter on the same subject make one wonder whether the sale of diathermy machines and other electrical apparatus should not be controlled by the State. It is high time for the profession to consider the matter seriously.

Yours, etc.,

JOHN HOETS.

143, Macquarie Street,
Sydney,
August 9, 1932.

"AVERTIN."

SIR: Having just completed a series of one hundred operations performed under "Avertin" anæsthesia, I thought your readers might find some observations on these cases of interest.

The majority of the series (78) consisted of simple major operations such as appendicectomy, radical cure for inguinal hernia, hysteropexy, salpingectomy and oophorectomy. The remainder included the following: Hysterectomy, (total and supravaginal) nephropexy, pyelolithotomy, prostatectomy, coccygectomy, excision of breast, elevation of depressed fracture of skull, mastoidectomy, removal of malignant growth of face with plastic repair, and enterectomy. Post-anæsthetic vomiting occurred only in six patients, and in all these vomiting had been a prominent feature before operation.

One death occurred and this, as proved by a *post mortem* examination, was due purely to a surgical condition and could not possibly be attributed to the anæsthetic used.

The youngest patient was eighteen months, and the oldest sixty-four years.

"Avertin" was used purely as a basal anæsthetic (0·1 gramme per kilogram of body weight) and was invariably supplemented by "open ether".

Veronal (seven and a half grains) was given the night before operation on every occasion.

In no case was the "Avertin" insufficient to induce deep sleep. In only two cases was this condition delayed for more than thirty minutes after administration, it being fifty minutes before complete anæsthesia occurred. In the first this was due to an error of dosage, the obstetric dose (0·075 gramme per kilogram of body weight) being given. In the second the patient was an excitable Italian, definitely a hyperthyreoid type.

In only two cases was it possible to make the skin incision without deepening the anæsthesia by administration of ether.

In one case only anxiety was caused by the condition of the patient during anæsthesia. This was a male, aged fifty, and weighing six stone, in an advanced stage of cachexia.

During the removal of a septic appendix he collapsed and the operation was completed as quickly as possible. A continuous rectal saline with 5% glucose was given as well as adrenaline hypodermically. Six hours later the patient's condition was satisfactory, and the convalescence was uneventful. He is now (six months later) nine stone and in perfect health. I can only explain this case by inferring that the appendix caused a toxic condition of the liver which, therefore, did not eliminate the "Avertin" sufficiently quickly.

Every patient who had previously had a general anæsthetic declared that "Avertin" was more pleasant both in the pre- and post-anæsthetic stage.

In operations on the head, the small amount of ether used, and the greater freedom of space for the surgeon, appealed to me as a distinct advantage.

My conclusions are as follows:

1. "Avertin" has a great advantage over solely ether or chloroform anæsthesia from the patient's point of view. The "psychological scar" left by the whole performance is immeasurably more satisfactory, and I have frequently heard patients remark they will never dread an operation as they did before.

2. "Avertin" makes the surgeon's task easier during operations on the head, where no anæsthetic machine is available, and the open method must be used.

3. By making the two days immediately succeeding operation less stormy, it saves the surgeon as well as the patient much worry.

4. The contraindications of this method are very few, the most important being impaired hepatic function. If this is borne in mind and the technique is carried out carefully, "Avertin" carries no added risk.

During my first few experiences with this mode of anæsthesia I felt very disappointed that it was necessary to use ether in addition, and I looked forward to the time when, after some practice, I could use morphine or "Narcophyn" to induce full anæsthesia. This temptation, which, I suppose, has assailed others, should, I think, be strongly resisted. It seems to me that only when full anæsthesia is induced by injection can the charge of uncontrolled anæsthesia, a term commonly used by your numerous correspondents, be justifiably applied.

As a matter of fact, if, after administration of "Avertin" only, I could make the skin incision without the patient feeling it, I would be much more alarmed than pleased.

Douglas Miller, in your last issue, paints a dramatic picture of the "pallid form" raising itself as if in vengeance, when the surgeon makes his incision in the insufficiently anæsthetized body, and upsetting the instruments on the floor. I would suggest to Dr. Miller that if he asks the anæsthetist to pinch the patient's arm and retain his instruments until this simple test does not evoke a response, he will save himself this distressing ordeal. Such a charge against "Avertin" is obviously unjustified. The anæsthetist, not the anæsthetic, is to blame.

The extremely divergent views as shown in the correspondence columns of your journal on this mode of anæsthesia seem incomprehensible to me, and I strongly suspect that large numbers of the correspondents have never seen "Avertin" used.

Yours, etc.,

S. GOLDBERG, M.B., Ch.M. (Sydney).

Banna Avenue,
Griffith,
New South Wales,
August 1, 1932.

POLIOMYELITIS.

SIR: In your issue of August 27 Dr. Goldberg questions the value of an increased cell content of the cerebro-spinal fluid as a diagnostic sign in the preparalytic stage of poliomyelitis. He brings forward in support of his argument statistics published by Dr. Steigrad, who found that of ninety cases "eight, that is, 9%, showed less than 4% of cells in the cerebro-spinal fluid". (Presumably this means less than four cells, and 4% has been written in error.) I should like to point out that Dr. Steigrad immediately follows his statement by the explanation that these normal cell counts were obtained in examinations of the cerebro-spinal fluid "done late in the disease, or particularly early in a very virulent form". Information gained from microscopical examination of *post mortem* material not available when Dr. Steigrad wrote his paper has eliminated from the final number of cases of poliomyelitis those with a normal cell count and suspected to have been a virulent form of that disease. In the remainder of Dr. Steigrad's eight cases the cell count was done as late as three or four weeks after paralysis had appeared, when normal figures are found in about 98% of cases. Obviously, these statistics are of no value in discussing the preparalytic stage.

In the 1931-1932 epidemic in New South Wales every case of poliomyelitis which occurred in the metropolitan area and was examined in the preparalytic stage had an increased cell content of the cerebro-spinal fluid in that stage, and no case in which a provisional diagnosis of poliomyelitis was excluded by a normal cell content later developed paralysis. The published records of cases of preparalytic poliomyelitis, such as those of W. L. Aycock (221 cases), Jean Macnamara (133 cases), and the Medical Research Committee of the University of Manitoba (161 cases), show that an increased cell content was always present. It is apparently a most constant and reliable

sign. The diagnosis of poliomyelitis cannot be considered definite in a case which has a normal cell content of the cerebro-spinal fluid in the preparalytic stage and paralysis of both legs which "disappeared completely in thirty-six hours". Claims of cure in doubtful cases are of no assistance in arriving at a correct estimation of the value of convalescent serum therapy and only promulgate inaccurate ideas as to its usefulness.

Yours, etc.,

KAREN HELMS.

135, Macquarie Street,
Sydney,
September 3, 1932.

Obituary.

WILLIAM MAYNE CLAYTON.

WE regret to announce the death of Dr. William Mayne Clayton, which occurred on September 9, 1932, at Hawthorn, Victoria.

NOTICE.

THE third annual reunion of the Royal Prince Alfred Hospital Resident and Ex-Resident Medical Officers' Association will be held from Monday, September 26, to Saturday, October 1, 1932, inclusive. A full programme of clinical and social functions has been arranged. Ex-resident medical officers of the hospital who are not already members of the Association and who are desirous of attending the reunion, can obtain full particulars from the Joint Honorary Secretaries at the Royal Prince Alfred Hospital, Sydney.

Books Received.

LIFE OF MENDEL, by Hugo Iltis. Translated by Eden and Cedar Paul; 1932. London: George Allen and Unwin Limited. Demy 8vo., pp. 294, with 10 illustrations and 12 plates. Price: 8s. 6d. net.

Diary for the Month.

SEPT. 20.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 21.—Western Australian Branch, B.M.A.: Branch.
SEPT. 23.—Queensland Branch, B.M.A.: Council.
SEPT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Victorian Branch, B.M.A.: Council.
SEPT. 29.—South Australian Branch, B.M.A.: Branch.
SEPT. 29.—New South Wales Branch, B.M.A.: Branch.
OCT. 4.—New South Wales Branch, B.M.A.: Council.
OCT. 5.—Victorian Branch, B.M.A.: Branch.
OCT. 5.—Western Australian Branch, B.M.A.: Council.
OCT. 6.—South Australian Branch, B.M.A.: Council.
OCT. 7.—Queensland Branch, B.M.A.: Branch.
OCT. 7.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of Local Associations.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

ADELAIDE CHILDREN'S HOSPITAL (INCORPORATED), ADELAIDE, SOUTH AUSTRALIA: Medical Superintendent.

DEVON PUBLIC HOSPITAL, LATROBE, TASMANIA: Surgeon Superintendent.

LEWISHAM HOSPITAL, SYDNEY, NEW SOUTH WALES: Clinical Assistant.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Seven Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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